CLINICAL AND ECHOCARDIOGRAPHIC PROFILE OF HYPERTROPHIC CARDIOMYOPATHY

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DECLARATION BY THE CANDIDATE

I hereby declare that the thesis entitled "**CLINICAL AND ECHOCARDIOGRAPHIC PROFILE OF HYPERTROPHIC CARDIOMYOPATHY**" submitted towards partial fulfillment of the requirements for the award of DM in Cardiology degree to BFUHS is a compilation of original work carried out by me under the supervision of Dr. Shibba Takkar Chhabra, Professor, Department of Cardiology and Dr. Abhishek Goyal, Associate Professor, Department of Cardiology. No part of this thesis has formed the basis for the award of any degree previously.

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Dedicated

to

my Family

&

Teachers

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“As we express our gratitude, we must never forget that the highest appreciation is not to utter words, but to live by them.”

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Dr. Anshuman Gupta

LIST OF ABBREVIATIONS

AICD	Automatic implantable cardioverter defibrillator
CMR	Cardiac magnetic resonance
CVA	Cardiovascular accident
DBP	Diastolic blood pressure
DM	Diabetes mellitus
DT	Deceleration time
ECG	Electrocardiography
ECHO	Echocardiography
EF	Ejection fraction
ESM	Ejection systolic murmur
F	Female
GCS	Global circumferential strain
GLS	Global longitudinal strain
HCM	Hypertrophic cardiomyopathy
HF	Heart failure
HTN	Hypertension
IPD	In patient department
IVRT	Isovolumic relaxation time
IVS-D	Interventricular septal thickness in diastole
IVS-S	Interventricular septal thickness in systole
LA	Left atrium
LAEF	Left atrial ejection fraction
LAVI	Left atrial volume index
LGE	Late gadolinium enhacement
LV	Left ventricle
LVH	Left ventricular hypertrophy
LVID-D	Left ventricular internal diameter in diastole
LVID-S	Left ventricular internal diameter in systole
LVOT	Left ventricular outflow tract
LVPW-D	Left ventricular posterior wall thickness in diastole
LVPW-S	Left ventricular posterior wall thickness in systole
M	Male

MPI	Myocardial performance index
MR	Mitral regurgitation
OPD	Out patient department
OSA	Obstructive sleep apnea
PAC’s	Premature atrial complexes
PASP	Pulmonary artery systolic pressure
PVC’S	Premature ventricular complexes
SAM	Systolic anterior motion of anterior mitral leaflet
SBP	Systolic blood pressure
SCD	Sudden cardiac death
SOB	Shortness of breath

ABSTRACT

Introduction: The Indian population exhibits a great range of geographic and genetic diversity in comparison to other populations; therefore there is need to examine the epidemiological and clinical presentation based on the gender specific differences, presenting complaints, age at presentation, family history.

Aims & Objectives:

1. To study the clinical and demographical profile of hypertrophic cardiomyopathy in our patients.
2. To compare echocardiographic characteristics with respect to subtypes of hypertrophic cardiomyopathy in our patients.

Material & Methods: This prospective, observational and non-blinded study was conducted to know the demographical and clinical profile of our hypertrophic cardiomyopathy patients. All patients who already presented between 1st march 2019 to 28th February 2020 and all follow up and new patients presented to DMCH Ludhiana between 1st march 2020 to 31st may 2021 were enrolled for study after taking informed and written consent.

Results: Out of total 103 patients of HCM, most of the patients had age group >60years i.e, 41.7%. Mean age of presentation was 56.28 ±13.93years. Overall the most common chief complaint was SOB in total of 80 (77.67%) patients. Past history of HTN was present in 71.84% (n=74) of patients. Family history of SCD was found in 21.29% (n=23) of patients. LVH was found in 48.54% (n=50). Giant T wave inversion was most commonly was found in apical (46%) type of HCM. LVH was found most commonly in apical (38%) type of HCM. MR with SAM was found in a total of 24 (23.3%) patients.

Conclusion: HCM is predominant in male sex with sex ratio being 2.1:1 in male and female respectively. Most common presenting complaint in both male and female sex was shortness of breath. Overall most common predominant type is reverse curvature (31.1%) type of HCM. Females had smaller left ventricular cavity and showed significant difference from male patients. Most common genetic mutation found to be was MYH7 and MYBPC3 i.e, total of 11 (22.19%) and 10 (20.8%) patients respectively were positive out of total 48 genetic tests done. Nonobstructive type of HCM was more common than obstructive type.

Keywords: HCM, genetic mutation, reverse curvature, obstructive and nonobstructive.

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INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is a genetically determined heart muscle disease caused by mutations in one of several sarcomere genes which encode components of the contractile apparatus.

HCM is characterized by an enormous diversity in both phenotypic expression and clinical course. The location, pattern, and extent of left ventricular hypertrophy (LVH) are heterogeneous, although the most common location for increased wall thickness is the basal anterior septum in continuity with the anterior free wall. HCM patients can develop one or more of the following morphologic abnormalities:

- LV outflow tract obstruction
- Diastolic dysfunction
- Myocardial ischemia
- Mitral regurgitation
- Systolic dysfunction (i.e., Left ventricular EF<50 percent)

These structural and functional abnormalities can produce a variety of symptoms, including:

- Fatigue
- Dyspnea
- Chest pain
- Palpitations
- Presyncope or syncope

In broad terms, the symptoms related to HCM can be categorized as those related to heart failure (HF), chest pain, or arrhythmias. Patients with HCM have an increased incidence of both supraventricular and ventricular arrhythmias and are at an increased risk for sudden cardiac death (SCD).

For the majority of patients with HCM, LVH is not progressive, and the clinical course is relatively benign. A small subset of patients, however, will progress to an advanced form of the disease that is characterized by systolic dysfunction with other adverse LV remodeling such as LV dilation and wall thinning present in some patients as well. Such patients are managed according to the standard approach to patients with HF due to systolic dysfunction.

Hypertrophy and fibrosis — HCM can present in infancy and childhood, but more commonly left ventricular hypertrophy (LVH) develops during the adolescent period[1]. In the majority of HCM patients, LV wall thickness measurements do not typically change once early adulthood is reached.

A multitude of pathologic studies in children and young adults with HCM who died suddenly demonstrated that increased wall thickness is due to myocyte hypertrophy and an expanded extracellular matrix composed of interstitial and replacement fibrosis[2]. The mechanisms leading to increased collagen matrix in HCM are not well defined but may be mediated by angiotensin II, an observation supported by studies using genetic mouse models of HCM in which increased interstitial fibrosis was attenuated by an angiotensin II receptor blocker[3].

The presence of late gadolinium enhancement (LGE) on contrast-enhanced cardiac magnetic resonance (CMR) imaging represents myocardial fibrosis.

Late onset disease — Although most HCM disease expression develops during childhood and adolescence, late-onset disease is recognized (i.e., LVH developing between ages 20 and 50 years) and was first noted in patients with mutations in cardiac myosin binding protein-C. Most cross-sectional studies of families with mutations in the cardiac myosin binding protein-C gene, which account for approximately 15 percent of cases of

familial HCM, have shown that the proportion of proven carriers of the mutation who have hypertrophy increases with age[4-7].

In contrast to familial early-onset HCM (which results from defects in the beta myosin heavy chain, cardiac troponin T, and alpha tropomyosin genes in more than 45 percent of cases), elderly-onset HCM is in some series associated with mutations in the cardiac myosin binding protein-C, troponin I, and alpha myosin heavy chain genes[7]. In addition, the likelihood of identifying any mutation may be lower in late onset HCM[8].

Additionally, HCM patients who are identified late in life (age >60 years) are at low risk for HCM-related death or adverse events compared with early-onset disease.

In a small subset of patients, sigmoid septal morphology (with maximal LV wall thickness ≥15 mm) is part of the phenotypic spectrum of HCM. A sigmoid shaped basal septum can also be observed in elderly individuals without HCM, but in these cases the maximal wall thickness of the septum is normal. Among HCM patients, this morphologic subtype appears to be less commonly associated with a mutation than other morphologic variants.

HCM with LV systolic dysfunction (ejection fraction <50 percent) — A small proportion of patients with HCM (<5 to 8 percent) eventually progress to a stage of disease associated with adverse left ventricular (LV) remodeling with reduced systolic performance (EF<50 percent). In some patients, there is cavity dilation and wall thinning due to a process of extensive myocardial fibrosis, as demonstrated by histologic examination in hearts removed at time of heart transplant and on cardiac magnetic resonance imaging with extensive late gadolinium enhancement[9-16]. Not all patients with HCM and LV systolic dysfunction will demonstrate cavity

dilation or wall thinning, and therefore the unifying definition is considered when a patient achieves an EF<50 percent.

This phase, previously termed "end-stage" or "burned out" HCM, is best described as "HCM with LV systolic dysfunction." HCM with LV systolic dysfunction is associated with a marked increase in the risk of SCD[15,16]. The mechanism of LV dysfunction is uncertain, but diffuse myocardial ischemia due to microvascular dysfunction leading to a process of substantial cell death and repair process in the form of myocardial replacement fibrosis may be important[17].

Nonobstructive with preserved systolic function (ejection fraction ≥50 percent) — The clinical profile of advanced HF in HCM also includes nonobstructive patients (i.e, patients with HCM but no significant Left ventricular outflow tract (LVOT) obstruction) with preserved systolic function (EF≥50percent) without significant LV remodeling, with average LV wall thicknesses of 22 mm, normal LV cavity dimensions, and usually very little myocardial fibrosis on contrast-enhanced cardiac magnetic resonance imaging[18]. The cause for HF in this subgroup of patients appears to be LV diastolic dysfunction due to impaired filling resulting in a low-output HF state. The time period between onset of symptoms and listing for heart transplant is on average eight years, underscoring the importance of close longitudinal follow-up in nonobstructive patients with HCM and HF symptoms and a low threshold to pursue cardiovascular evaluation for heart transplantation once symptoms becomes substantial.

HCM Variants:

1. Mid-cavity obstructive HCM[19]
2. Mid-cavity obstruction with LV apical aneurysm[20]
3. Apical HCM[21]
4. Right ventricular obstruction
5. Obstructive HCM in elderly adults: Sigmoid septal morphology[22]

6. Transition to end-stage HCM[23]

Echocardiographic HCM Variants[24]:

1. Reverse curvature
2. Sigmoid
3. Neutral
4. Apical
5. Apical with mid segment involvement (Mixed)

SYMPTOMS: The majority of clinically identified patients with HCM have no or minor symptoms[25]. Thus, affected children and adolescents are often diagnosed during family screening. Patients who are asymptomatic, or have minor symptoms, have a better prognosis than those with more severe symptoms.

Patients with mild to moderate limitation may have a stable clinical course with effective therapy, or may experience slow progression of symptoms with advancing age. Typical symptoms include dyspnea, chest pain, syncope, and palpitations.

The major disease-related complications of HCM are ventricular arrhythmias leading to sudden death, chest pain, progressive heart failure symptoms or HF death, atrial arrhythmias including atrial fibrillation, and embolic stroke.

Mortality -In series published in the 1980s, the annual mortality of patients with HCM in referral center populations was 4 to 6 percent per year. The major causes of death in HCM are sudden cardiac death, HF, and stroke.

Risk factors -Patients with HCM have an increased risk of death from several causes (SCD, HF, and stroke). Various risk factors are age at diagnosis, female gender, symptom status, obstruction, coronary disease

and stress induced ischemia, heart failure, arrythmias, stroke, obesity and infective endocarditis.

AIMS AND OBJECTIVES

- To study the clinical and demographical profile of hypertrophic cardiomyopathy in our patients.
- To compare echocardiographic characteristics with respect to subtypes of hypertrophic cardiomyopathy in our patients.

REVIEW OF LITERATURE

Tanjore RR et al in 2006 reported an increased male preponderance, the sex ratio M:F 3.7:1 and the age at onset was found to be more than a decade early in familial cases (30 ± 10 yrs) compared to non familial cases (44 ± 14 yrs)[26].

Study by Rangaraju A et al in 2014 revealed 37% of sporadic cases and 63% of familial HCM cases, with a sex ratio of 3.1:1, indicating a male preponderance. The mean age at onset of 38 years was observed irrespective of the gender. Interestingly, males were predominant in the age group of 21-40 years exhibiting non-obstructive type of HCM while female preponderance was observed in the age group of 0-20 years and 40-60 years with high incidence of obstructive HCM cases associated with severe clinical manifestations and poor prognosis, based on the significant echocardiographic parameters[27].

Biswas A et al in 2015 concluded that there were 59 patients with a clinical diagnosis of HCM with a preponderance of disease in males with a ratio (men, women) of 5.5:1. Average age of onset of the disease was late 30 s (39.2 ± 14.5) with familial HCM accounting for 18% (n = 9) for total HCM families (n = 50). Nonobstructive kind of HCM was more prevalent as compared to obstructive HCM (66.1% vs. 33.9%). Average posterior wall LV thickness of the HCM patients was 16 ± 4.8 mm and IVS thickness was 21 ± 8.3 mm with familial patients having greater wall thickness as compared to sporadic patients. Sequencing of hotspot region of MYH7 identified three mutations in three different patients in Indian population[28].

Marian A Jet al in 2017 reported that mutations in over a dozen genes encoding sarcomere associated proteins cause HCM. MYH7 and MYBPC3, encoding β-myosin heavy chain and myosin-binding protein C, respectively, are the two most common genes involved, together

accounting for ≈50% of the HCM families. In ≈40% of HCM patients, the causal genes remain to be identified[29].

Luo Q et al in 2020 divided a total of 90 HCM patients with HTN and 172 patients without HTN into a hypertensive group and non-hypertensive group. The clinical characteristics, cardiac structure and function, and prognosis of the two groups were compared and found that HCM patients with HTN had fewer syncope events in their medical histories (8% vs. 22%, $P < 0.01$) and sudden deaths in the family (3% vs. 10%, $P < 0.05$). The prevalence of apical hypertrophy (18% vs. 7%, $P < 0.01$) and midventricular obstruction (26% vs. 15%, $P < 0.05$) was higher in the HTN group[30].

A study was conducted by Tandon R et al in 2020 where a total of 233 HCM patients were taken. The mean age of the patients was 53 ± 14.5 years with male preponderance (70%). Thirty six percent patients were asymptomatic, 37% mildly symptomatic (in class I-II), and 27% severely symptomatic (in class III-IV). Patients were classified into six phenotypes consisting of five classically described phenotypes and one atypical phenotype. Reverse curvature phenotype was most prevalent (49%) and symptomatic phenotype with 19.3% having significant resting left ventricular outflow tract (LVOT) obstruction. Other phenotypes were 19% apical mid, 11% neutral, 6% sigmoid, and 4% atypical. Male:female ratio for reverse curvature phenotype was 3:1, apical (mid) phenotype 2.4:1, and both apical and neutral phenotypes 2:1. Sigmoid phenotype had female to male ratio of 2.5:1[31].

Inki Moon et al in 2020 reported the prevalence of HCM steadily increased by 1.9-fold from 0.016% (n = 6313) in 2010 to 0.031% (n = 13,035) in 2016. A gradual increase in the prevalence of HCM was consistently observed in both genders, with male predominance during the entire study period. The ratio of males to females was approximately 2:1 with no changes during the study period. The prevalence of HCM increased very

slowly in patients aged between 20 and 59 years, whereas the prevalence sharply increased in patients aged over 60 years. HCM prevalence was highest in patients aged between 70 and 79 years. In all age categories except the age category of ≥80 years, male HCM had a numerically higher prevalence than female HCM with a male to female ratio of 2–4:1. HCM prevalence was similar in the age category of ≥80 years. The mean age of patients with HCM remained relatively stable from 60.7±12.9 years in 2010 to 62.8±13.2 in 2016. Of note, female patients with HCM were older by approximately 8 years than their male counterparts throughout the study period. In 2016, the most common clinical presentation was chest pain (n = 5763 [44.2%]), followed by dyspnea (n = 1313 [10.1%]). Only 361 patients (2.8%) presented with syncope in 2016. Clinical presentation with dyspnea showed an increasing trend from 8.7% in 2010 to 10.1% in 2016. Hypertension was the most common comorbidity in patients with HCM, followed by dyslipidemia. The proportion of patients with heart failure (HF) significantly increased from 15.3% in 2010 to 38.5% in 2016 ($p<0.001$)[32].

Erica Zaiser et al in 2020 interviewed the HCM patients and the symptoms most commonly reported was tiredness (89%), shortness of breath (89%), shortness of breath with physical activity (89%), and dizziness/light-headedness (89%). Other symptoms commonly reported included chest pain (angina) (70%), chest pain (angina) with physical exertion (70%), and palpitations (fluttering or rapid heartbeat) (81%)[33].

Luis R. Lopes et al in 2021 based on echocardiographic parameter reported a total of 34 individuals out of total 29826 had minimum left ventricular wall thickness (MLVWT) of 15 mm or more, for a prevalence of 0.11% (95% CI, 0.08%-0.15%); the prevalence was 0.22% (95% CI, 0.14%-0.30%) in male participants and 0.04% (95% CI, 0.01%-0.06%) in female participants. With a cutoff value of 13 mm or more, the prevalence was 0.96% (95% CI, 0.85%-1.07%) overall, 2.07% (95% CI, 1.82%-

2.32%) in male participants, and 0.12% (95% CI, 0.07%-0.18%) in female participants[34].

A study was conducted by Trongtorsak A. et al in 2021 where female gender was significantly associated with an increased risk of all-cause mortality (pooled OR = 1.63, 95% CI: 1.26–2.10, $p \leq 0.001$), HCM-related mortality (pooled OR = 1.47, 95% CI: 1.08–2.01, $p = 0.015$), and worsening HF or HF hospitalization (pooled OR = 2.05, 95% CI: 1.76–2.39, $p \leq 0.001$)[35].

MATERIALS AND METHODS

This prospective, observational and non-blinded study was conducted to know the demographical and clinical profile of our hypertrophic cardiomyopathy patients. All patients who already presented between 1st march 2019 to 28th February 2020 and all follow up and new patient's presented to DMCH Ludhiana between 1st march 2020 to 31st may 2021 were enrolled for study after taking informed and written consent.

Source of data:

Data collection was done by analyzing the patients with clinical features of HCM and confirmation on echocardiography.

Method of collection of data

All enrolled patients underwent:

1. ECG
2. ECHO
3. BNP levels (if available)
4. Holter (if indicated)
5. Routine haemogram

Family members were screened for HCM by ECG and Echocardiography. The American College of Cardiology (ACC)/American Heart Association (AHA) consensus guidelines recommend 12 years as the starting age for family screening of first-degree relatives of affected probands with hypertrophic cardiomyopathy (HCM).

Inclusion Criteria

Any patient who had presented to DMCH/HDHI in patient / out patient clinic with echocardiographic diagnosis of HCM.

Exclusion criteria

1. Aortic valve stenosis
2. Physiologic athlete's heart

Proposed Clinical Family Screening Strategies

With Echocardiography or Cardiovascular Magnetic Resonance (and 12-Lead Electrocardiography) for Detection of HCM with Left Ventricular Hypertrophy[36].

Age <12 years: Optional unless:

1. Malignant family history of premature death from HCM, or other adverse complications.
2. Competitive athlete in an intense training program.
3. Onset of symptoms.
4. Other clinical suspicion of early left ventricular hypertrophy.

Age 12 to 21 years: Every 12 to 18 months.

Age >21 years: Imaging at onset of symptoms, or possibly at 5-year intervals at least through midlife; more frequent intervals for imaging are appropriate in families with malignant clinical course or history of late-onset HCM.

Genetic Basis and Testing

HCM is transmitted as a mendelian trait with an autosomal dominant pattern of inheritance; every offspring of an affected relative has a 50% chance of inheriting the disease

HCM is known to be caused by mutations in 11 or more genes encoding proteins of the thick and thin contractile myofilament components of the cardiac sarcomere or the adjacent Z-disc. Two sarcomere genes, β-myosin heavy chain (MYH7) and myosin-binding protein C (MYBPC3) are by far the most common, accounting for 70% of patients for whom

genotyping has been successful. Troponin T (TNNT2), troponin I (TNNI3), and several other genes are each responsible for 5% or fewer cases.

Five milliliter of intravenous blood sample was collected with a prior written consent if required in this study. Genomic DNA will be extracted using phenol‑chloroform method [37] for genetic testing. Sequencing of hotspot region (exon 23) of MYH7 gene for variant identification using Sanger method will be done, if required.

Echocardiographic criteria for diagnosis of HCM[38]

The echocardiographic examination should include:

- Confirming LV hypertrophy.
- Assessment of LVOT obstruction.
- Systolic anterior motion – SAM.
- Assessment of systolic and diastolic LV function and left atrial size.

The following two-dimensional (2D) echocardiographic criteria are used to aid diagnosis:

1. Unexplained maximal wall thickness >15mm in any myocardial segment, or
2. Septal/posterior wall thickness ratio >1.3 in normotensive patients, or
3. Septal/posterior wall thickness ratio >1.5 in hypertensive patients.

Up to one third of patients with HCM will have obstruction under basal (resting) conditions (defined as gradients ≥30mm Hg).

Another one third will have labile, physiologically provoked gradients (<30mm Hg at rest and ≥30mm Hg with physiologic provocation).

The final one third will have the non-obstructive form of HCM (gradients <30mm Hg at rest and with provocation). Marked gradients ≥50mm Hg, either at rest or with provocation, represent the conventional threshold for

surgical or percutaneous intervention if symptoms cannot be controlled with medications.

Echocardiographic predictors of unfavourable outcome

- Sudden death: Maximal wall thickness >30mm.
- HCM related death: LVOT gradient at rest >30mmHg, EF<50%.
- All-cause mortality: Left atrial diameter >48mm.
- Heart failure development: LA volume >27ml/m^2 at baseline or during follow up.
- Intraventricular dyssynchrony: >45msec

Electrocardiographic features for diagnosis of HCM

- Left atrial enlargement.
- Left ventricular hypertrophy with associated ST segment / T-wave abnormalities.
- Deep, narrow (“dagger-like”) Q waves in the lateral > inferior leads.
- Giant precordial T-wave inversions in apical HCM.
- Signs of WPW (short PR, delta wave).

Dysrhythmias: atrial fibrillation, supraventricular tachycardias, premature atrial complexes (PAC’s), premature ventricular complexes (PVC’s), ventricular tachycardia.

Definitions

1. Family history: A record of the relationships among family members along with their medical histories which includes current and past illnesses and may show a pattern of certain diseases in a family.
2. Sudden cardiac death (SCD): Sudden cardiac death (SCD) is an unexpected death due to cardiac causes that occurs in a short time period (generally within 1 hour of symptom onset) in a person with known or unknown cardiac disease.

Choice of patients for genetic analysis

In this study patients were chosen for genetic analysis on the basis of strong family history of similar disease, history of SCD in family and younger age of presentation of symptoms.

Statistical Analysis

Data were described in terms of range; mean ±standard deviation (± SD), frequencies (number of cases) and relative frequencies (percentages) as appropriate. To determine whether the data were normally distributed, a Kolmogorov-Smirnov test was used. Comparison of quantitative variables between the study groups was done using Student t-test and Mann Whitney *U* test for independent samples for parametric and non-parametric data respectively. For comparing categorical data, Chi square ($\chi 2$) test was performed and exact test was used when the expected frequency is less than 5. A probability value (*p* value) less than 0.05 was considered statistically significant. All statistical calculations were done using (Statistical Package for the Social Science) SPSS 21version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows.

Limitation of this study

Due to covid-19 pandemic, follow up of the patients was hampered and less number presented for follow up.

OBSERVATIONS AND RESULTS

During the prospective study period from 1st march 2020 to 31st may 2021 a consecutive of n=103 patients were taken in for study. Out of 103 patients, 27 were admitted (IPD) and 76 patients were taken as outdoor (OPD) clinic basis.

Out of 27 admitted patients, 9 were already on follow up and rest 18 were admitted first time.

Out of 76 OPD patients, 22 were already on follow up and rest 54 presented first time.

Genetic analysis was done in a total of 48 patients (18 were follow up on OPD basis and 30 were admitted first time) out of a total of 78.

TABLE 1: Age and gender wise distribution of HCM patients

		Sex				Total	Chi-square value	p-value
		F		M				
Age group	< 50	5	15.2%	25	35.7%	30	4.594	0.032
	51-60	12	36.4%	18	25.7%	30	1.232	0.267
	> 60	16	48.5%	27	38.6%	43	0.906	0.341
Total		33	100.0%	70	100.0%	103		

Out of total 103 patients of HCM, most of the patients had age group >60years i.e, 41.7% (38.6% M and 48.5% F) where as in <50years of age group there were 29.12% (35.7% M and 15.2% F) patients.

Rest 29.12% (25.7% M and 36.4% F) of patients were between 51-60years age group.

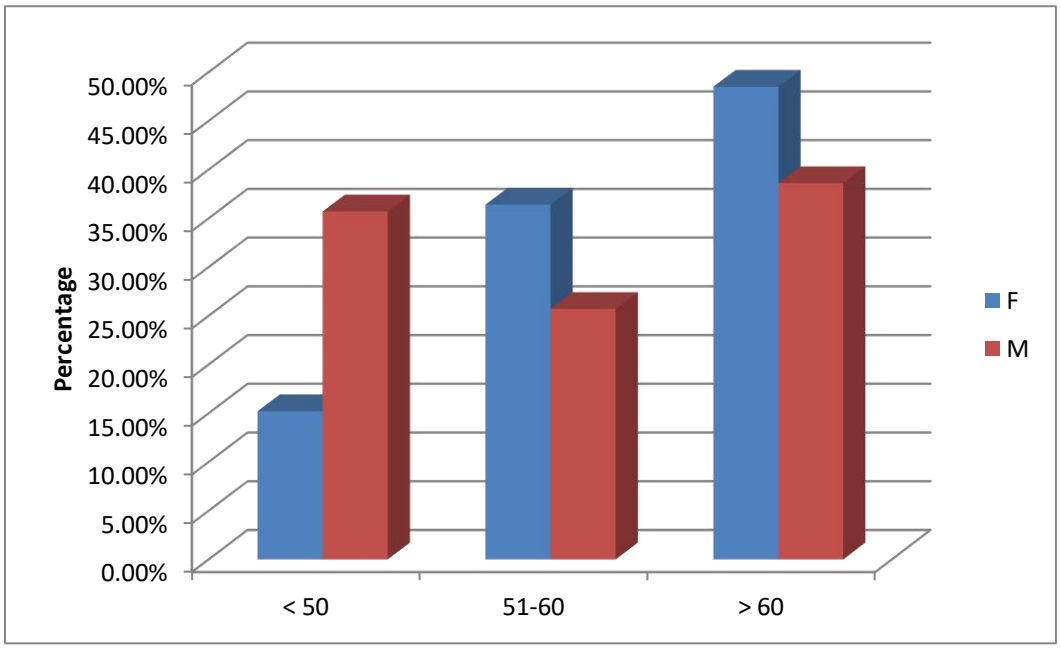

Figure 1: Age and gender wise distribution of HCM patients

Table 2a: Mean age of presentation in HCM patients

	N	Minimum	Maximum	Mean	Std. Deviation
Age	103	17.00	90.00	56.28	13.93

Out of total 103 patients, minimum and maximum age of the patient was 17years and 90years respectively with mean age of 56.28 ±13.93years.

Table 2b: Gender wise distribution of HCM patients according to mean age of presentation

	F		M		Z	p-value
	Mean	SD	Mean	SD		
Age	59.70	12.87	54.67	14.20	1.725	0.088

Comparing gender wise, mean age of presentation in males was 54.67±14.20years whereas in females was 59.70±12.87years.

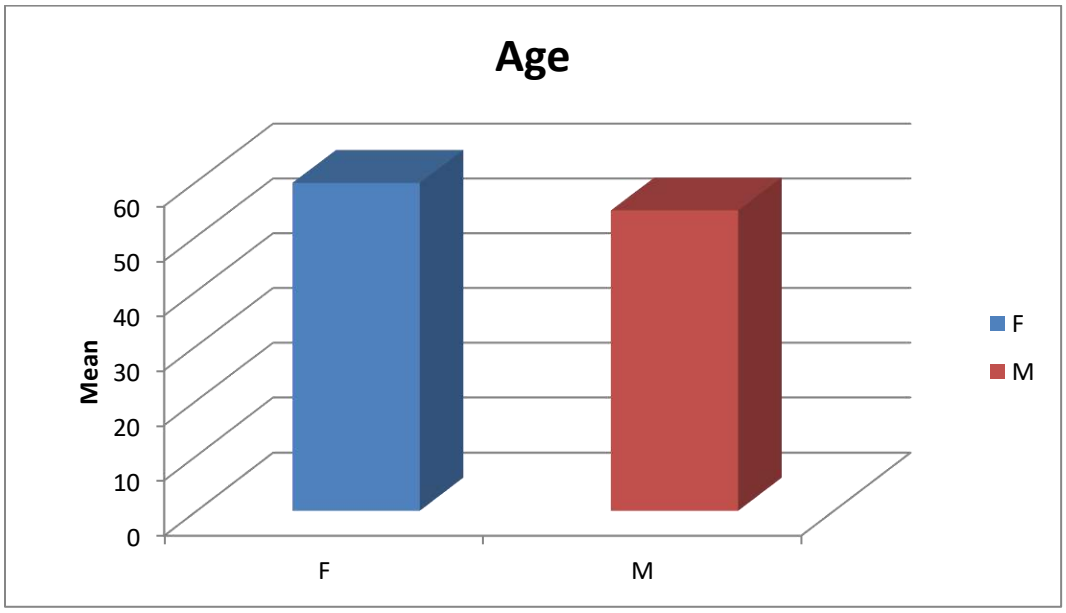

Figure 2: Gender wise distribution of HCM patients according to mean age of presentation

Table 3: Gender wise distribution of HCM patients according to the chief complaints

Chief complaints		Sex				Total	Chi-square value	p-value
		F		M				
Chest pain	No	27	81.8%	60	85.7%	87	0.259	0.610
	Yes	6	18.2%	10	14.3%	16		
SOB	No	6	18.2%	17	24.3%	23	0.482	0.488
	Yes	27	81.8%	53	75.7%	80		
Palpitations	No	25	75.8%	63	90.0%	88	3.656	0.056
	Yes	8	24.2%	7	10.0%	15		
Syncope	No	28	84.8%	63	90.0%	91	0.578	0.447
	Yes	5	15.2%	7	10.0%	12		
Presyncope	No	31	93.9%	66	94.3%	97	0.005	0.944
	Yes	2	6.1%	4	5.7%	6		

Overall the most common chief complaint was SOB in total of 80 (77.67%) patients (75.7% M and 81.8% F), followed by chest pain in 16 (15.53%) patients (14.3% M and 18.2% F), palpitations in 15 (14.56%) patients (10% M and 24.2% F), syncope in 12 (11.65%) patients (10% M and 15.2% F) and presyncope in 6 (5.82%) patients (5.7% M and 6.1% F).

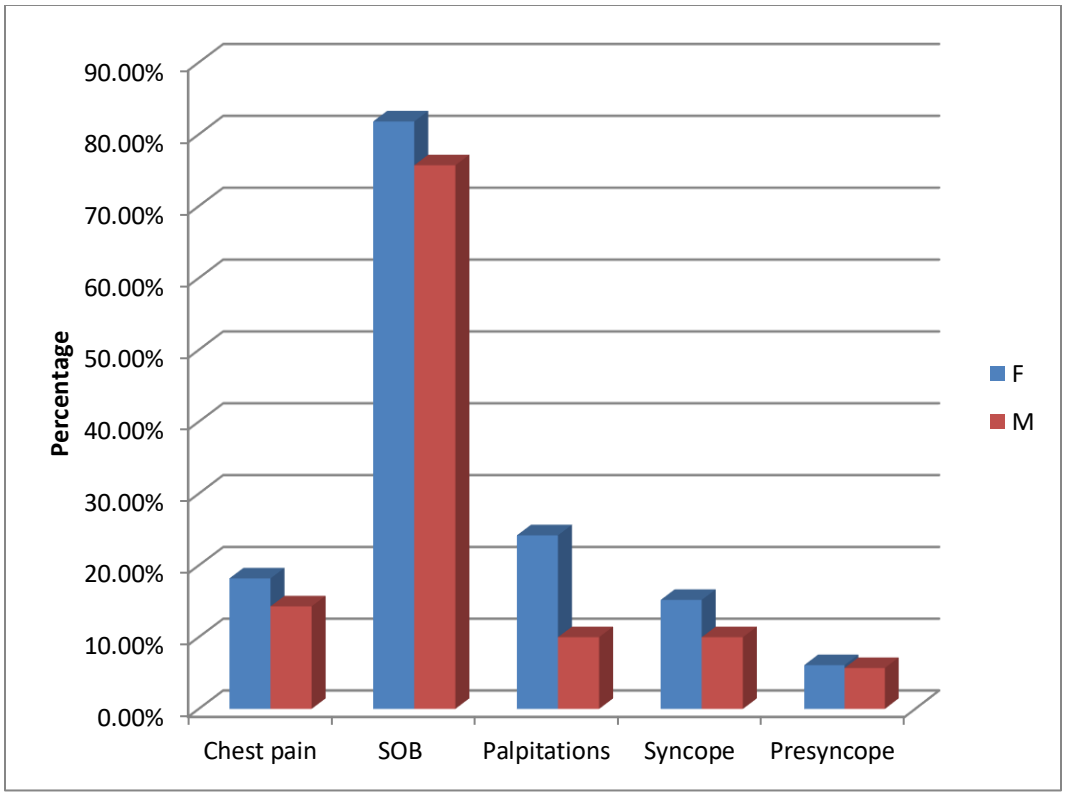

Figure 3: Gender wise distribution of HCM patients according to the chief complaints

Table 4: Gender wise distribution of HCM patients according to the type of HCM

		Sex				Total	Chi-square value	p-value
		F		M				
Type	Apical	6	18.2%	24	34.3%	30	2.818	0.093
	Mid and apical	5	15.2%	14	20.0%	19	0.350	0.786
	Neutral	1	3.0%	11	15.7%	12	3.505	0.097
	Reverse curvature	16	48.5%	16	22.9%	32	6.878	0.012
	Sigmoid	5	15.2%	5	7.1%	10	1.641	0.284
Total		33	100%	70	100%	103	11.602	0.021

Overall the most common type was reverse curvature in a total of 32 (31.1%) patients (22.9% M and 48.5% F), followed by apical in 30 (29.1%) patients (34.3% M and 18.2% F), mid and apical in 19 (18.4%) patients (20% M and 15.2% F), neutral in 12 (11.7%) patients (15.7% M and 3% F), sigmoid in 10 (9.7%) patients (7.1% M and 15.2% F).

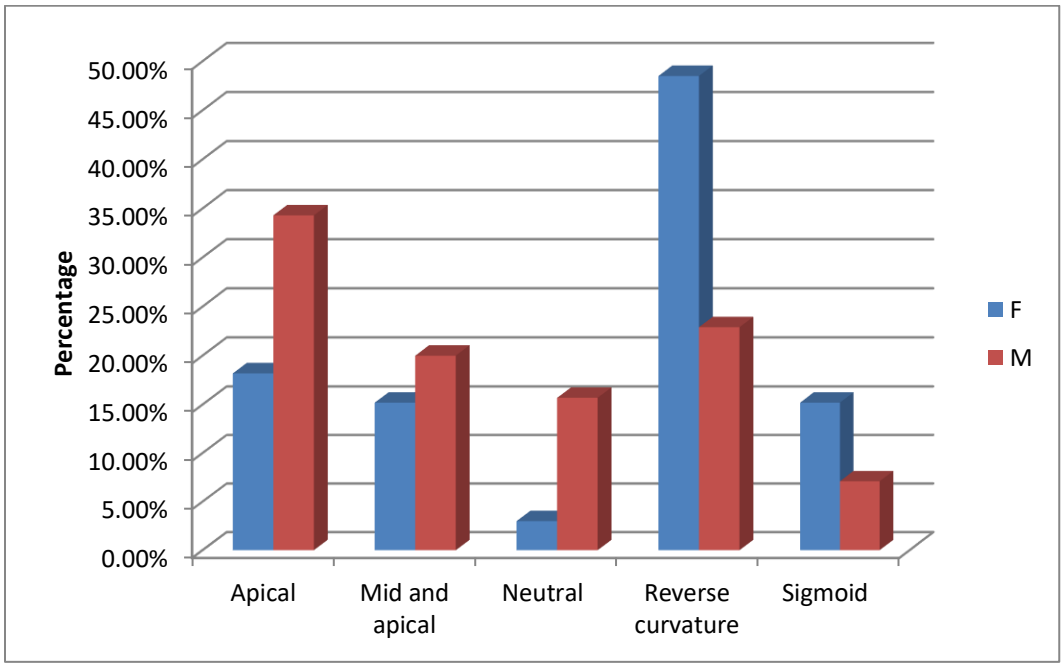

Figure 4: Gender wise distribution of HCM patients according to the type of HCM

Table 5: Gender wise distribution of HCM patients according to the past history

		Sex				Total	Chi-square value	p-value
Past history		F		M				
HTN	No	4	12.1%	25	35.7%	29	6.171	0.018
	Yes	29	87.9%	45	64.3%	74		
Bronchial Asthma	No	33	100.0%	70	100.0%	103		
Arrythmia	No	31	93.9%	66	94.3%	97	0.005	0.944
	Yes	2	6.1%	4	5.7%	6		
CVA	No	32	97.0%	69	98.6%	101	0.302	0.583
	Yes	1	3.0%	1	1.4%	2		
Diabetes mellitus	No	30	90.9%	69	98.6%	99	3.528	0.060
	Yes	3	9.1%	1	1.4%	4		
Heart failure	No	31	93.9%	70	100.0%	101	4.326	0.101
	Yes	2	6.1%	0	0.0%	2		
AICD implantation	No	33	100.0%	70	100.0%	103		

Overall out of total 103 patients, past history of HTN was present in 71.84% (n=74) of patients, arrhythmia in 5.82% (n=6), diabetes mellitus in 3.88% (n=4), heart failure and CVA in 1.85% (n=2).

Comparing gender wise, past history of HTN was present in 45 (64.3%) male and in 29 (87.9%) female patients. Past history of arrhythmia was found to be in 4 (5.7%) male and in 2 (6.1%) female patients. The arrhythmia found to be was atrial fibrillation.

Past history of bronchial asthma, CVA, diabetes mellitus, heart failure and AICD implantation was less common.

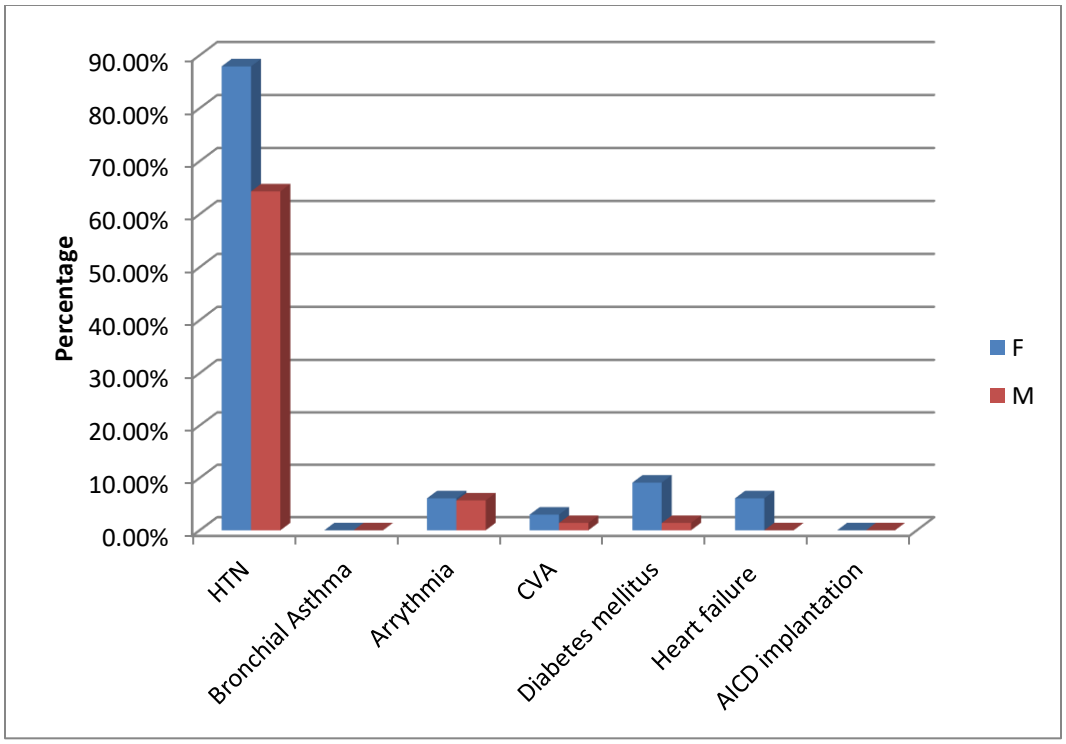

Figure 5: Gender wise distribution of HCM patients according to the past history

Table 6: Gender wise distribution of HCM patients according to the personal history

		Sex				Total	Chi-square value	p-value
Personal history		F		M				
Smoker/ Non Smoker	Non	33	100.0%	68	97.1%	101	0.962	0.327
	Yes	0	0.0%	2	2.9%	2		
Alcoholic/ Non-Alcoholic	Non	33	100.0%	63	90.0%	96	3.541	0.060
	Yes	0	0.0%	7	10.0%	7		
OSA	No	28	84.8%	64	91.4%	92	1.018	0.313
	Yes	5	15.2%	6	8.6%	11		
Marathon runner	No	33	100.0%	70	100.0%	103		

Overall out of total 103 patients, personal history of OSA was found in 10.67% (n=11) of patients, alcohol in 6.79% (n=7), smoking in 1.94% (n=2).

Comparing gender wise there were 2 (2.9%) male and none of the female patients who smoke. There were 7 (10%) male and none of the female patients who took alcohol. Personal history of OSA was in 6 (8.6%) of male and in 5 (15.2%) of female patients. None of the male and female patients had history of marathon running.

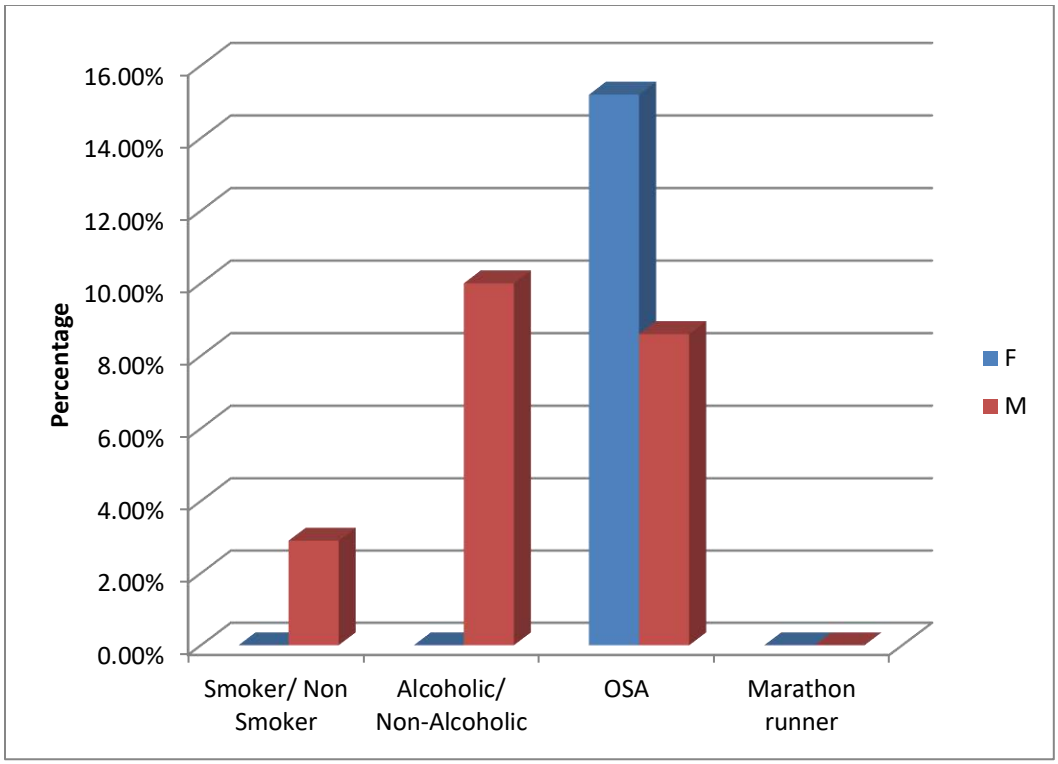

Figure 6: Gender wise distribution of HCM patients according to the personal history

Table 7: Gender wise distribution of HCM patients according to the family history of SCD

		Sex				Total	Chi-square value	p-value
		F		M				
Family History of SCD	No	24	72.7%	56	80.0%	80	0.684	0.408
	Yes	9	27.3%	14	20.0%	23		

Overall out of total 103 patients, family history of SCD was found in 21.29% (n=23) of patients.

Comparing gender wise family history of SCD was found in 14 (20%) male and 9 (27.3%) of female patients.

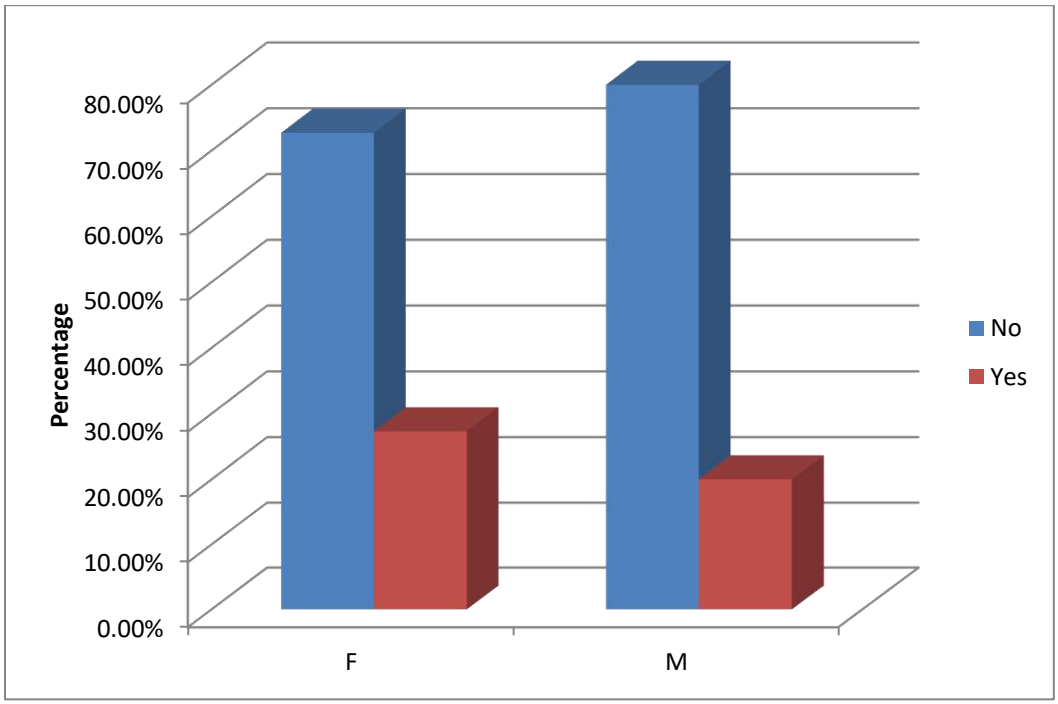

Figure 7: Gender wise distribution of HCM patients according to the family history of SCD

Table 8: Gender wise distribution of HCM patients according to the ECG characteristics

ECG characteristics		Sex				Total	Chi-square value	p-value
		F		M				
Axis	Left	5	15.2%	5	7.1%	10	1.641	0.200
	Normal (-30 to +100 degree)	28	84.8%	65	92.9%	93		
Rhythm	Irregularly irregular	3	9.1%	4	5.7%	7	4.838	0.089
	NSR	28	84.8%	66	94.3%	94		
	Pacemaker	2	6.1%	0	0.0%	2		
LVH (cornell voltage criteria)	No	18	54.5%	35	50.0%	53	2.227	0.328
	Yes	15	45.5%	35	50.0%	50		
QTc group	Normal (<460msec in males and <470msec in females)	12	36.4%	43	61.4%	55	5.662	0.021
	Prolonged (>460msec in males and >470msec in females)	21	63.6%	27	38.6%	48		
Giant T wave inversion (Negative T wave with voltage >10mm)	No	17	51.5%	36	51.4%	53	0.000	0.993
	Yes	16	48.5%	34	48.6%	50		

Overall out of 103 patients, normal ECG axis was found in 86.11% (n=93) and left axis deviation in 9.7% (n=10). Normal sinus rhythm (NSR) in 87.03% (n=94), irregularly irregular in 6.79% (n=7) and pacemaker rhythm in 1.85% (n=2). LVH was found in 48.54% (n=50). Prolonged QTc in 46.60% (n=48). Giant T wave inversion in 48.54% (n=50).

Comparing gender wise, left axis deviation in ECG was found in 5 (7.1%) male and in 5 (15.2%) of female patients where as others had normal axis. Irregularly irregular rhythm was found in 4 (5.7%) of male and 3 (9.1%) of female patients. LVH was found in 35 (50%) of male and 15 (45.5%) of female patients. Prolonged QTc was found in 27 (38.6%) of male and 21 (63.6%) of female patients. Giant T wave inversion was found in 34 (48.6%) of male and 16 (48.5%) of female patients.

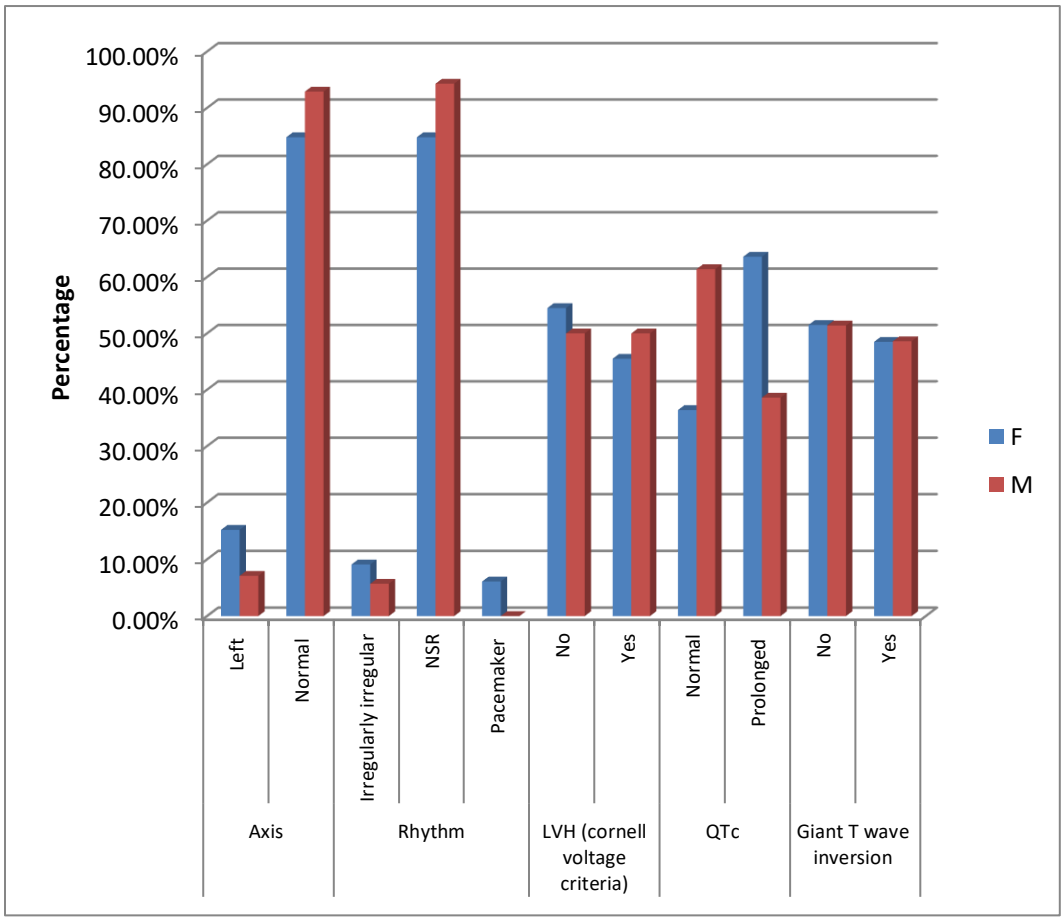

Figure 8: Gender wise distribution of HCM patients according to the ECG characteristics

Table 9a: Mean values of 12 lead ECG characteristics in HCM patients

12 Lead ECG characteristics	N	Minimum	Maximum	Mean	Std. Deviation
PR interval(ms)	96	100.00	200.00	157.50	22.34
QRS duration(ms)	103	80.00	200.00	117.48	11.78
QTc (Bazett's)(ms)	103	390.00	660.00	461.99	34.42

Out of total 103 patients, mean PR interval was 157.50±22.34msec.

Mean QRS duration was 117.48±11.78msec.

Mean QTc (Bazett's) was 461.99±34.42msec.

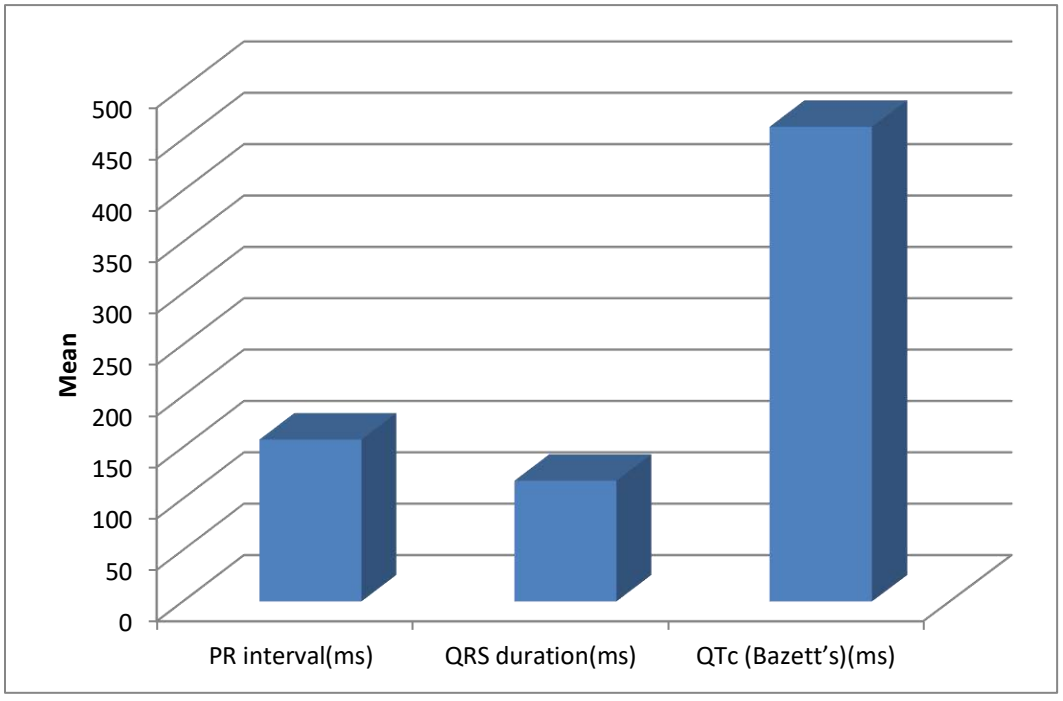

Figure 9a: Mean values of 12 lead ECG characteristics in HCM patients

Table 9b: Gender wise distribution of 12 lead ECG characteristics in HCM patients according to mean values

12 lead ECG characteristics	F		M		Z	p-value
	Mean	SD	Mean	SD		
PR interval(ms)	156.00	24.86	158.18	21.26	-0.442	0.660
QRS duration(ms)	119.70	18.28	116.43	6.82	1.319	0.190
QTc (Bazett's)(ms)	462.24	26.99	461.87	37.60	0.051	0.960

Comparing gender wise mean PR interval in males and females was 158.18±21.26msec and 156±24.86msec.

Mean QRS duration in males and females was 116.43±6.82msec and 119.70±18.28msec.

Mean QTc (Bazett's) in males and females was 461.87±37.60msec and 462.2±26.99msec.

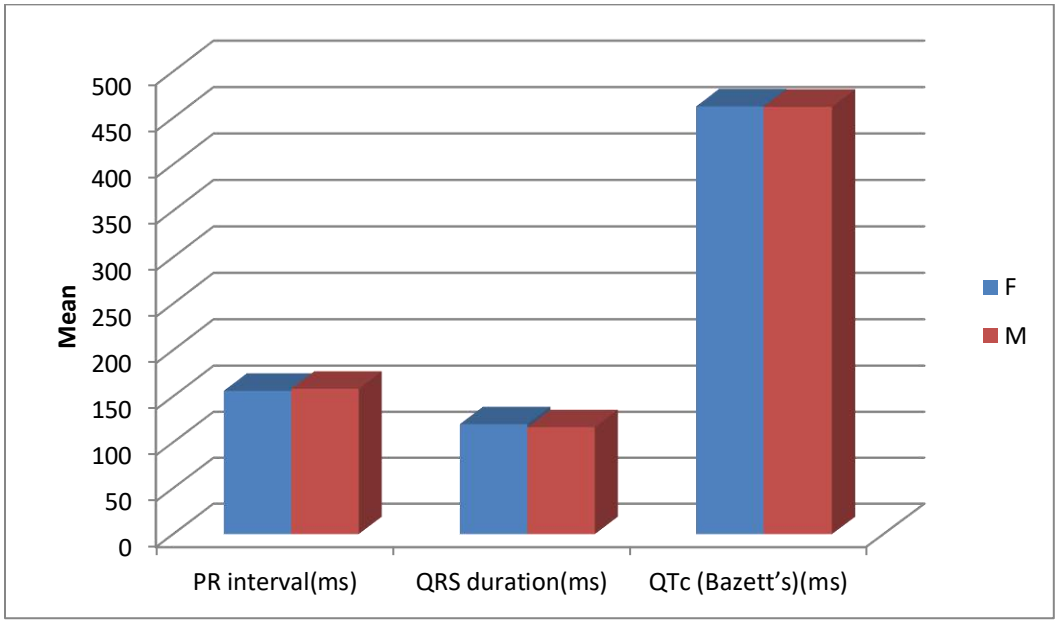

Figure 9b: Gender wise distribution of 12 lead ECG characteristics in HCM patients according to mean values

Table 10: Gender wise distribution of HCM patients according to the echocardiography characteristics

		Sex				Total	Chi-square value	p-value
Echo characteristics		F		M				
MR	No	13	39.4%	52	74.3%	65	11.727	0.001
	Yes	20	60.6%	18	25.7%	38		
SAM	No	17	51.5%	58	82.9%	75	11.130	0.001
	Yes	16	48.5%	12	17.1%	28		
Mid-cavity gradient	Absent	33	100.0%	64	91.4%	97	3.004	0.083
	Yes	0	0.0%	6	8.6%	6		
Total		33	100.0%	70	100.0%	103		

Overall out of total 103 patients, MR was found in 36.89% (n=38) of patients, SAM in 27.13% (n=28) and mid-cavity gradient in 5.82% (n=6).

Comparing gender wise, MR was found in 18 (25.7%) of male and 20 (60.6%) of male patients. SAM was found in 12 (17.1%) of male and 16 (48.5%) of female patients. Mid-cavity gradient was found in 6 (8.6%) of male and none of the female patients.

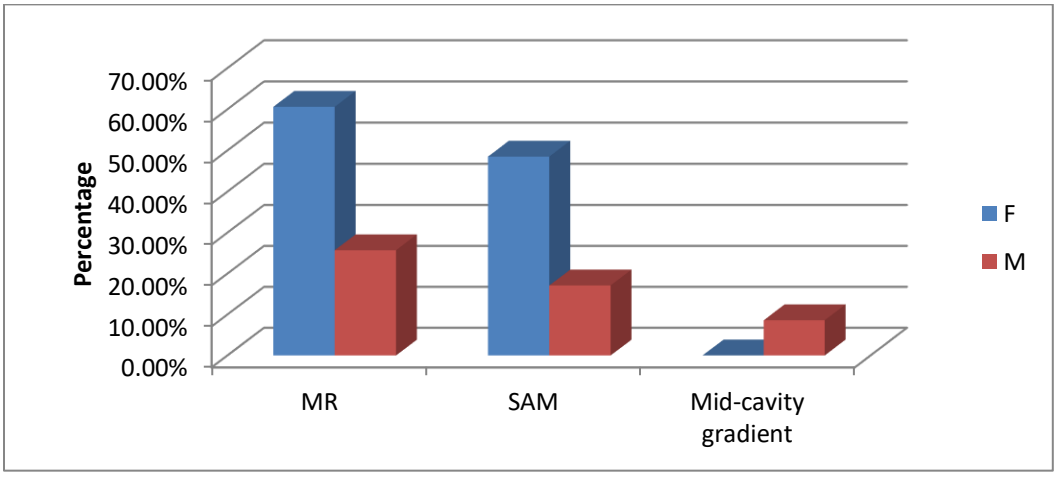

Figure 10: Gender wise distribution of HCM patients according to the echocardiography characteristics

Table 11: Gender wise distribution of HCM patients according to the mean echocardiographic values

Echocardiographic parameters	F		M		Z	p-value
	Mean	SD	Mean	SD		
Maximum wall thickness (mm)	25.52	4.87	24.33	3.48	1.414	0.161
Opposing wall ratio	2.43	1.07	1.93	0.89	2.167	0.013
LVID-S (mm)	26.12	5.17	30.06	6.19	-3.167	0.002
LVID-D (mm)	41.09	6.09	44.43	7.83	-2.157	0.033
LVPW-S(mm)	13.06	3.56	14.13	2.95	-1.596	0.114
LVPW-D (mm)	10.71	3.39	11.93	2.72	-1.953	0.054
IVS-S (mm)	24.09	6.69	20.76	6.26	2.467	0.015
IVS-D (mm)	21.58	6.16	18.39	5.90	2.533	0.013
LVOT gradient: resting (mm of Hg)	57.82	23.22	61.60	31.76	-0.356	0.725
Mitral valve- E/A	1.30	0.59	1.18	0.50	1.052	0.296
E/e’	18.26	5.91	15.24	5.31	2.603	0.011
IVRT	78.76	19.53	75.53	13.40	0.980	0.329
DT	183.58	49.71	177.59	45.14	0.608	0.544
PASP (mm of Hg)	33.52	10.49	30.41	9.13	1.533	0.128
LAVI(ml/m^2)	35.48	3.84	34.06	5.64	1.316	0.191
LAEF=(MaxV-MinV)/MaxV%	53.76	6.29	53.56	9.04	0.114	0.909
EF by 2D echo	57.61	6.81	59.83	1.43	-2.625	0.010
EF by 3D echo	57.70	6.51	59.86	1.90	-2.567	0.012
Strain derived EF	50.70	7.05	52.98	4.02	-2.085	0.040
Stroke volume(ml)	49.52	8.42	52.84	10.67	-1.574	0.119
LV MPI	0.52	0.07	0.50	0.08	1.152	0.252
GLS (- %)	14.71	4.05	14.15	3.85	0.667	0.506
GCS (- %)	16.45	4.04	16.80	4.87	-0.357	0.722
LA strain(peak) %	19.80	2.76	21.06	4.02	-1.626	0.107

Mean opposing wall ratio was 1.93±0.89 in male and 2.43±1.07 in female patients.

Mean LVID-S was 30.06±6.19mm in male and 26.12±5.17mm in female patients.

Mean LVID-D was 44.43±7.83mm in male and 41.09±6.09mm in female patients.
Mean IVS-S was 20.76±6.26mm in male and 24.09±6.69mm in female patients.
Mean IVS-D was 18.39±5.90mm in male and 21.58±6.16mm in female patients.
Mean E/e' ratio was 15.24±5.31 in male and 18.26±5.91 in female patients.

These all above parameters and mean EF by 2D, 3D and by strain derived echocardiography showed statistical difference where as all other parameters such as maximum wall thickness, LVPW-S and D, LVOT gradient resting and valsalva, mitral valve E/A, E/e', IVRT, DT, PASP, LAVI, LAEF, stroke volume, LV MPI, LA strain, GLS and GCS showed no statistical difference.

Table 12: Mean values of echocardiographic parameters in HCM patients

Echocardiographic parameters	N	Minimum	Maximum	Mean	Std. Deviation
Maximum wall thickness (mm)	103	15.00	38.00	24.71	3.99
Opposing wall ratio	74	1.23	4.22	2.34	0.70
LVID-S (mm)	103	16.00	50.00	28.80	6.14
LVID-D (mm)	103	23.00	59.00	43.36	7.46
LVPW-S (mm)	103	7.10	24.00	13.79	3.18
LVPW-D (mm)	103	6.50	22.00	11.54	2.99
IVS-S (mm)	103	10.00	40.00	21.83	6.56
IVS-D (mm)	103	8.00	32.00	19.41	6.14
LVOT gradient: resting (mm of Hg)	27	20.00	105.00	59.22	26.16
Mitral valve- E/A	98	0.50	2.80	1.22	0.53
E/e’	103	8.00	39.00	16.20	5.66
IVRT	103	32.00	113.00	76.56	15.60
DT	103	60.00	384.00	179.50	46.49
PASP (mm of Hg)	103	15.00	55.00	31.41	9.64
LAVI(ml/m^2)	103	19.00	50.00	34.51	5.16
LAEF=(MaxV-MinV)/MaxV%	103	22.00	73.00	53.62	8.23
EF by 2D echo	103	35.00	60.00	59.12	4.12
EF by 3D echo	103	35.00	65.00	59.17	4.09
Strain derived EF	103	32.00	60.00	52.25	5.26
Stroke volume(ml)	103	30.00	97.00	51.78	10.09
LVMPI	103	0.32	0.77	0.50	0.08
GLS (- %)	103	4.50	22.20	14.33	3.90
GCS (- %)	103	4.00	26.10	16.69	4.60

Mean maximum wall thickness was 24.71±3.99mm.

Mean opposing wall ratio was 2.34±0.70.

Mean LVID-S was 28.80±6.14mm.

Mean LVID-D was 43.36±7.46mm.

Mean LVPW-S was 13.79±3.18mm.

Mean LVPW-D was 11.54±2.99mm.

Mean IVS-S was 21.83±6.56mm.

Mean IVS-D was 19.41±6.14mm.

Mean LVOT gradient at rest was 59.22±26.16mm of Hg.

Mean mitral E/A was 1.22±0.53.

Mean mitral E/e' was 16.20±5.66.

Mean IVRT was 76.56±15.6msec.

Mean DT was 179.50±46.49msec.

Mean PASP was 31.41±9.64mm of Hg.

Mean LAVI was 34.51±5.16ml/m^2.

Mean LAEF was 53.62±8.23%.

Mean EF by 2D echo was 59.12±4.12%.

Mean EF by 3D echo was 59.17±4.09%.

Mean strain derived EF was 52.25±5.26%.

Mean stroke volume was 51.78±10.09ml.

Mean LVMPI was 0.50±0.08.

Mean GLS was -14.33±3.9%.

Mean GCS was -16.69±4.6%.

Table 13a: Mean values of pulse rate and blood pressure in HCM patients

General physical examination	N	Minimum	Maximum	Mean	Std. Deviation
Pulse(/min)	103	50.00	138.00	78.65	12.88
SBP (on medication)	103	88.00	180.00	130.70	16.22
DBP(on medication)	103	60.00	100.00	79.81	9.05

Out of total 103 patients, mean pulse rate was 78.65±12.88/min.

Mean SBP (on medication) was 130.70±16.22mm of Hg.

Mean DBP (on medication) was 79.81±9.05mm of Hg.

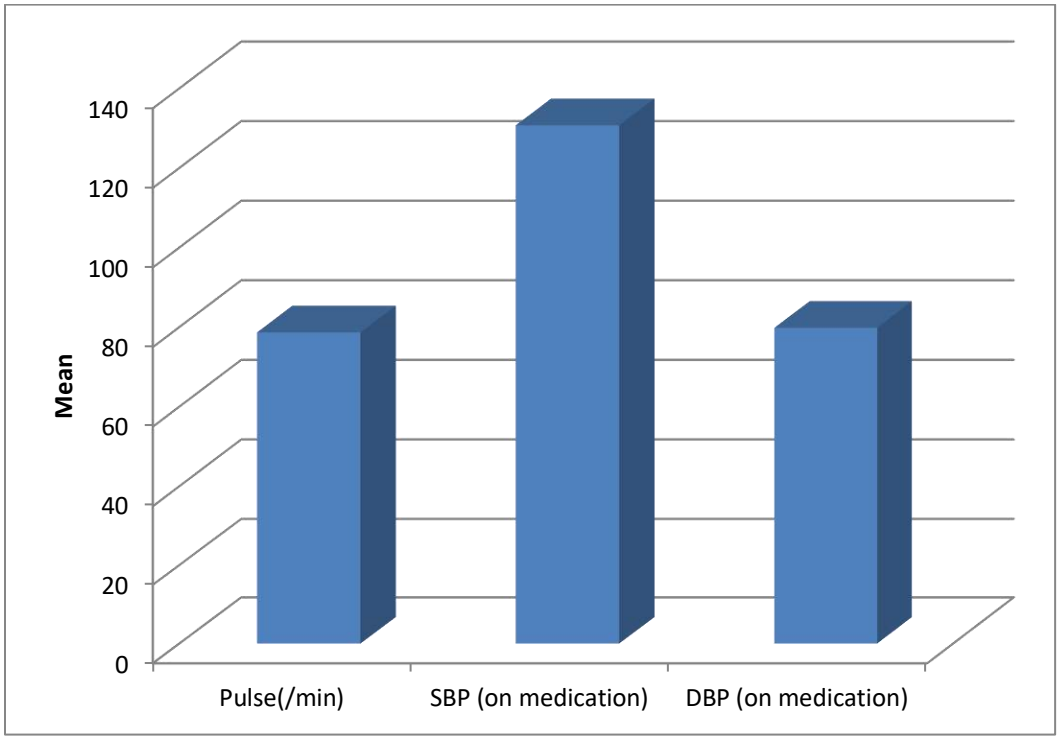

Figure 11a: Mean values of pulse rate and blood pressure in HCM patients

Table 13b: Gender wise distribution of HCM patients according to mean values of pulse rate and blood pressure

	F		M		Z	p-value
General physical examination	Mean	SD	Mean	SD		
Pulse(/min)	78.03	14.19	78.94	12.30	-0.334	0.739
SBP (on medication)	131.03	17.23	130.54	15.84	0.142	0.888
DBP (on medication)	78.91	9.04	80.23	9.09	-0.689	0.493

Comparing gender wise, mean pulse rate in males and females was 78.94±12.30/min and 78.03±14.19/min.

Mean SBP in males and females was 130.54±15.84mm of Hg and 131.03±17.23mm of Hg.

Mean DBP in males and females was 80.23±9.09mm of Hg and 78.91±9.04mm of Hg.

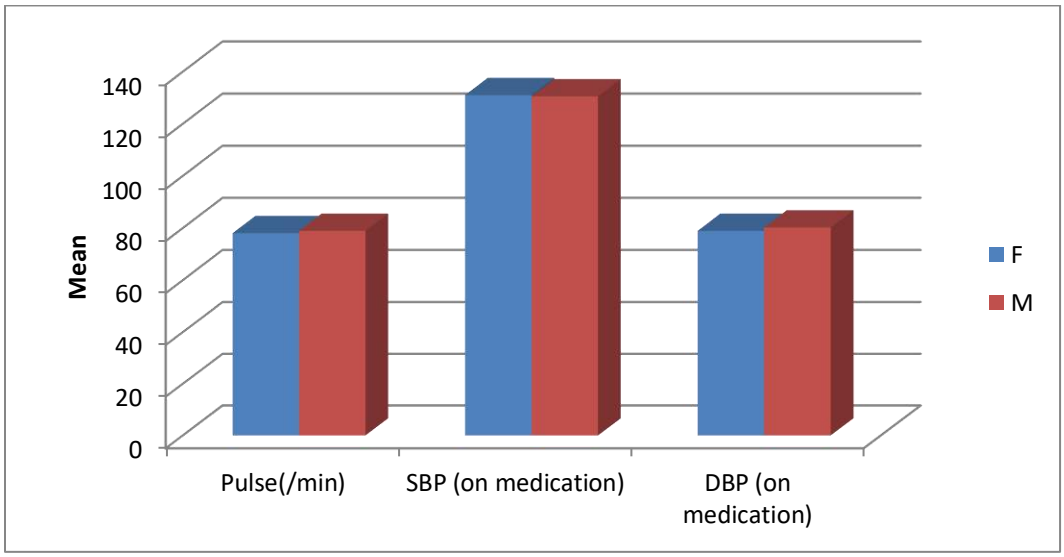

Figure 11b: Gender wise distribution of HCM patients according to mean values of pulse rate and blood pressure

Table 14: Comparison of giant T wave inversion in various types of HCM patients

	Giant T wave inversion		Total	Chi-square value	p-value
Type	No	Yes			
Apical	7	23	30	13.403	0.000
Mid and apical	7	12	19	1.992	0.206
Neutral	8	4	12	1.258	0.360
Reverse curvature	21	11	32	3.731	0.059
Sigmoid	10	0	10	10.448	0.001
Total	53	50	103		

Giant T wave inversion was found in total 50 (48.54%) out of total 103 patients and most commonly was found in apical (46%) type of HCM.

Less commonly was found in other types like mid and apical (24%), reverse curvature (22%), neutral (8%) and sigmoid variety.

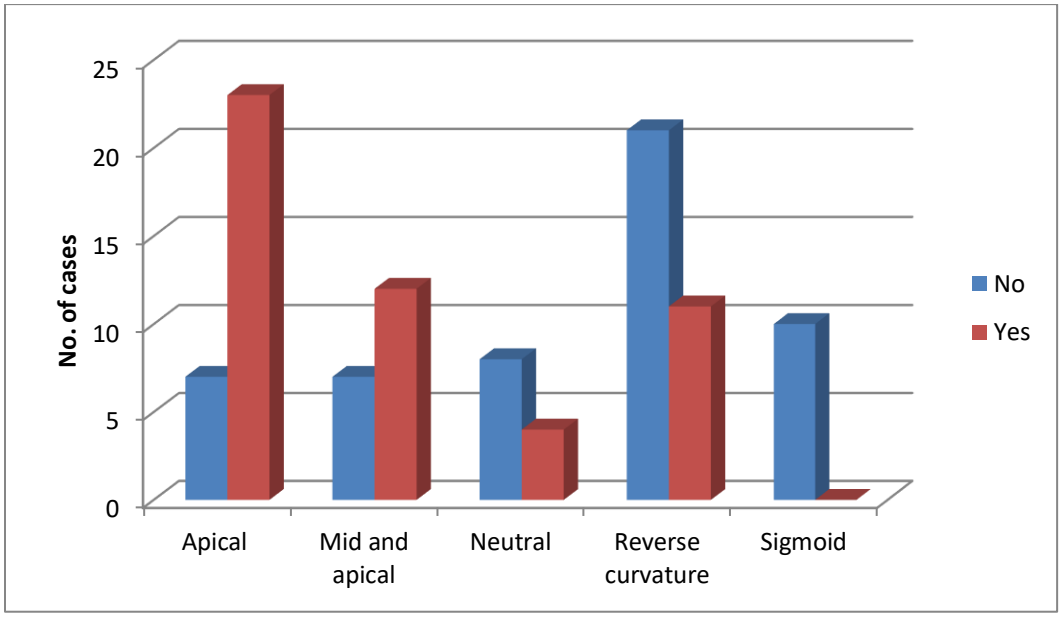

Figure 12: Comparison of giant T wave inversion in various types of HCM patients

Table 15: Comparison of LVH in various types of HCM patients

Type	LVH		Total	Chi-square value	p-value
	No	Yes			
Apical	11	19	30	3.707	0.082
Mid and apical	8	11	19	0.816	0.449
Neutral	8	4	12	1.258	0.360
Reverse curvature	18	14	32	0.427	0.531
Sigmoid	8	2	10	3.612	0.094
Total	53	50	103		

LVH was found in total 50 (48.54%) out of total 103 patients and most commonly was found in apical (38%) type of HCM.

Less commonly was found in other types like reverse curvature (28%), mid and apical (22%), neutral (8%) and sigmoid (4%) variety.

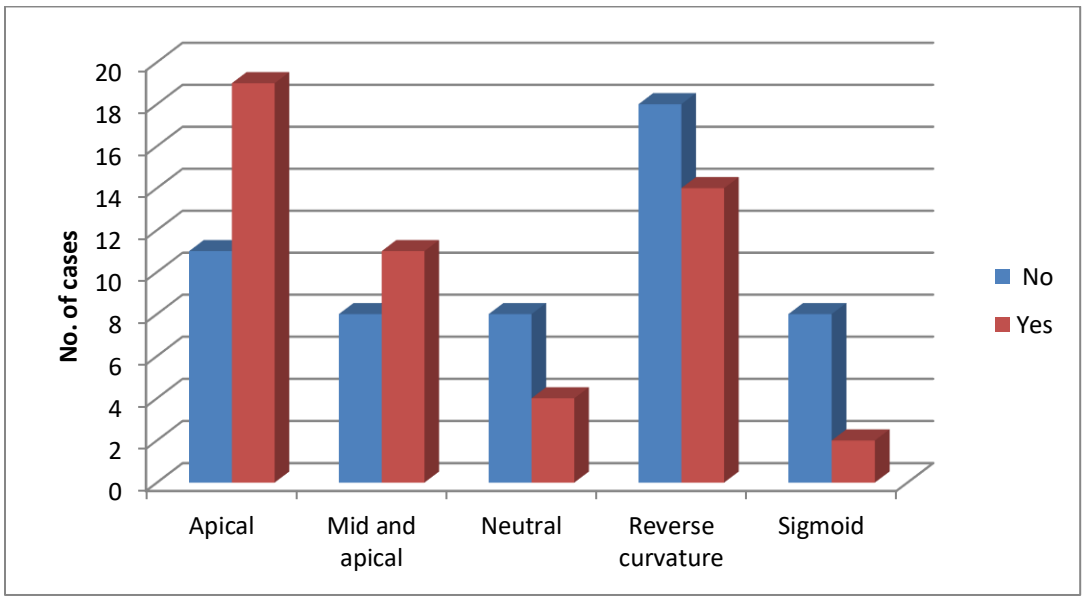

Figure 13: Comparison of LVH in various types of HCM patients

Table 16: Comparison of SAM in various types of HCM patients

Type	SAM		Total	Chi-square value	p-value
	No	Yes			
Apical	29	1	30	12.165	0.000
Mid and apical	18	1	19	5.656	0.017
Neutral	12	0	12	5.076	0.033
Reverse curvature	10	22	32	40.158	0.000
Sigmoid	6	4	10	0.919	0.338
Total	75	28	103		

SAM was found in total 28 (27.18%) out of total 103 patients and most commonly was found in reverse curvature (78.57%) type of HCM.

Less commonly was found in other types like sigmoid (14.28%), apical (3.6%), mid and apical (3.6%).

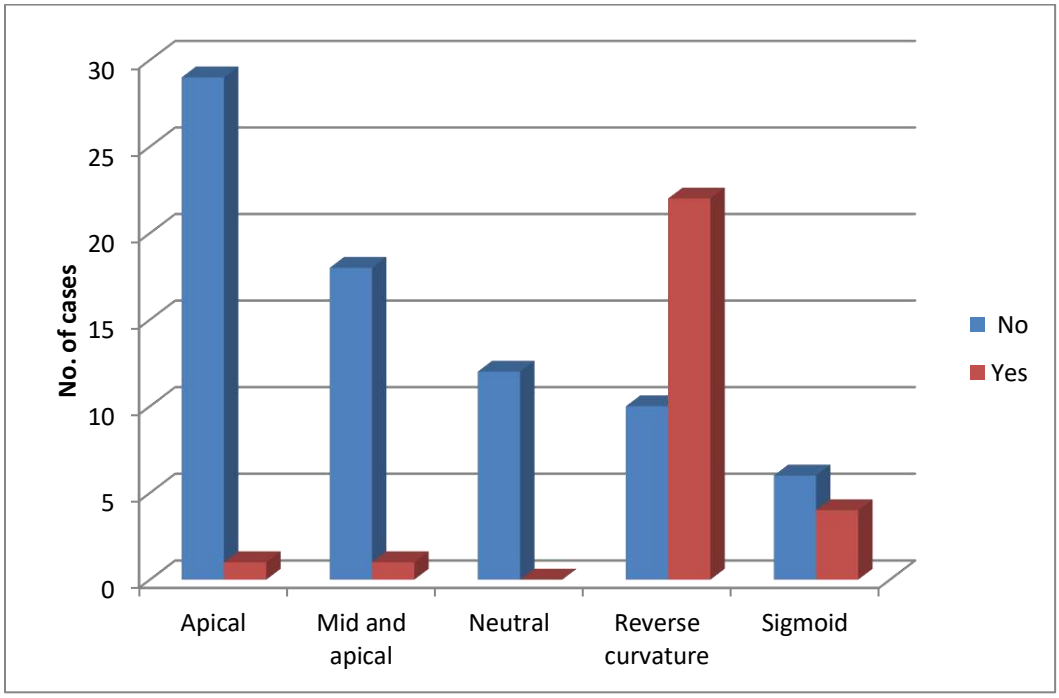

Figure 14: Comparison of SAM in various types of HCM patients

Table 17: Comparison of MR in various types of HCM patients

	MR		Total	Chi-square value	p-value
Type	No	Yes			
Apical	22	8	30	1.901	0.186
Mid and apical	14	5	19	1.120	0.430
Neutral	11	1	12	4.758	0.052
Reverse curvature	12	20	32	13.074	0.000
Sigmoid	6	4	10	0.046	0.830
Total	65	38	103		

MR was found in total 38 (36.9%) out of total 103 patients and most commonly was found in reverse curvature (52.6%) type.

MR found in other types like apical (21%), mid and apical (13.16%), sigmoid (10.5%) and neutral (2.6%) variants was mild and not due to SAM.

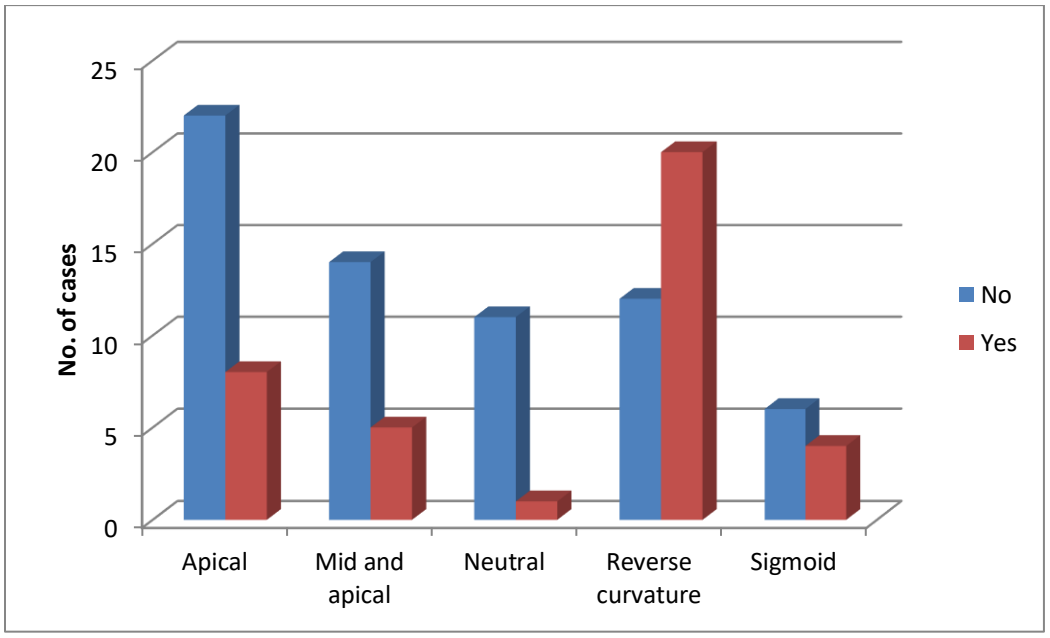

Figure 15: Comparison of MR in various types of HCM patients

Table 18: Comparison of SAM with MR in various types of HCM patients

SAM			MR		Total	Chi-square value	p-value
			No	Yes			
No	Type	Apical	22	7	29	0.932	0.334
		Mid and apical	14	4	18	0.197	0.657
		Neutral	11	1	12	1.005	0.316
		Reverse curvature	9	1	10	0.571	0.677
		Sigmoid	5	1	6	0.017	0.896
	Total		61	14	75		
Yes	Type	Apical	0	1	1	0.173	0.678
		Mid and apical	0	1	1	0.173	0.678
		Reverse curvature	3	19	22	0.035	0.851
		Sigmoid	1	3	4	0.438	0.508
	Total		4	24	28		

MR with SAM was found in a total of 24 (23.3%) patients out of 103 patients. Most commonly it was found in reverse curvature type of HCM in a total of 19 (79.17%) patients followed by less commonly in sigmoid (14.29%), mid and apical (4.2%), apical (4.2%) variants.

MR without SAM was found in a total of 14 (13.6%) patients out of 103 patients. MR associated with apical, mid and apical, neutral variants were mild and not related to SAM.

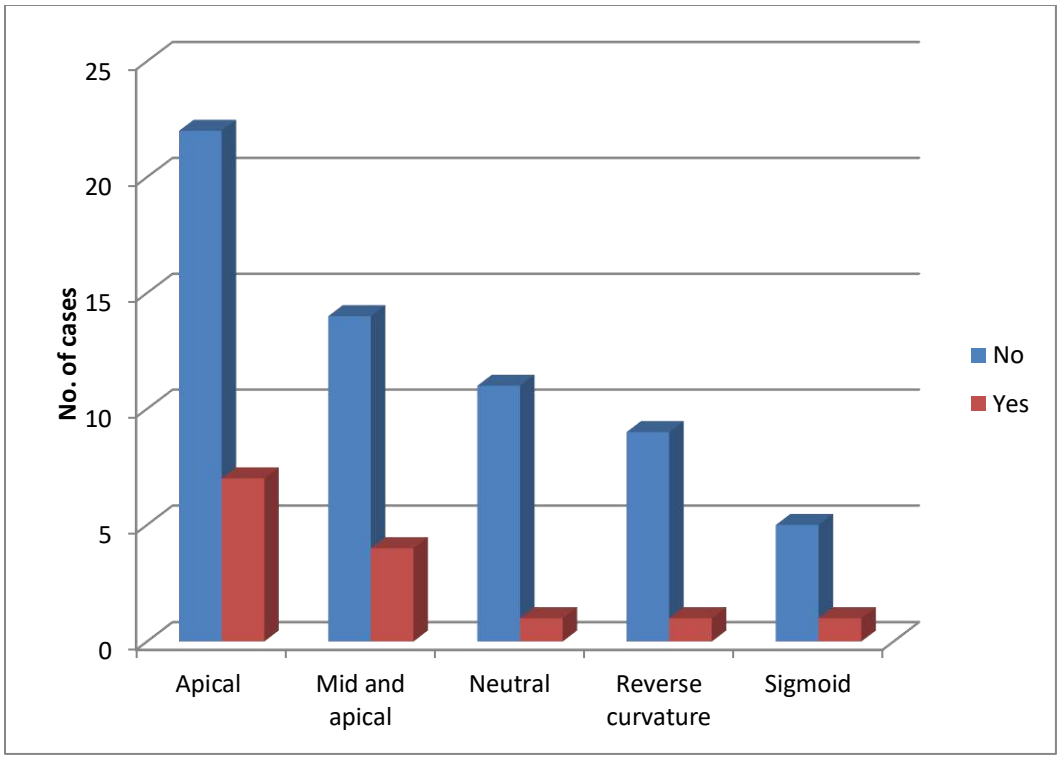

Figure 16a: Comparison of MR without SAM in various types of HCM patients

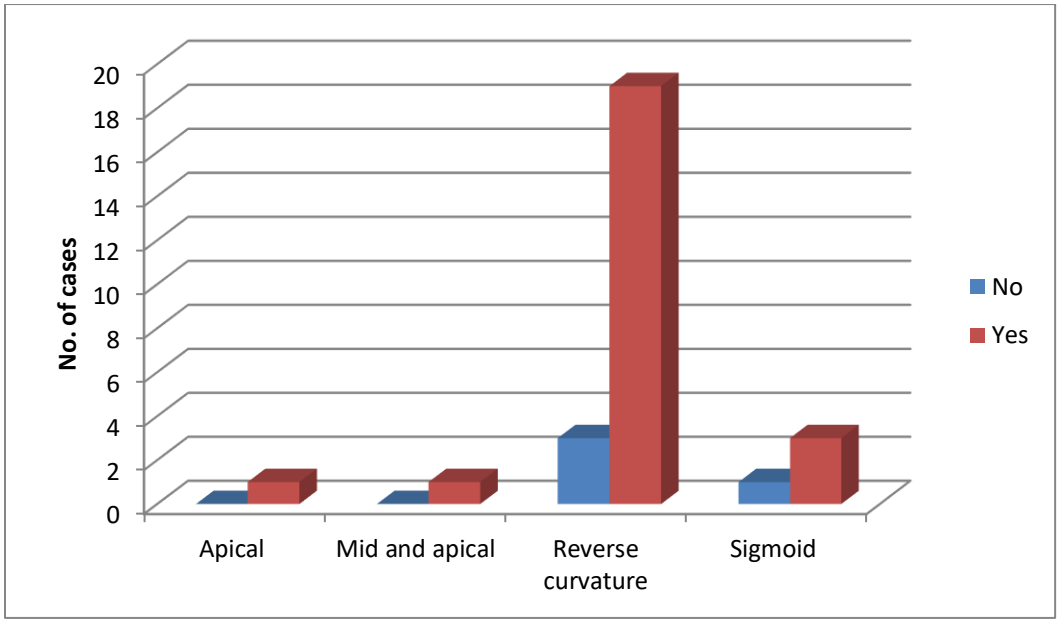

Figure 16b: Comparison of MR with SAM in various types of HCM patients

Table 19: Comparison of mid-cavity gradient in various types of HCM patients

Type	Mid-cavity gradient		Total	Chi-square value	p-value
	Absent	Yes			
Apical	29	1	30	0.479	0.489
Mid and apical	15	4	19	9.847	0.010
Neutral	12	0	12	0.840	0.359
Reverse curvature	31	1	32	0.617	0.663
Sigmoid	10	0	10	0.685	0.408
Total	97	6	103		

Mid-cavity gradient was found in total 6 (5.8%) out of total 103 patients and most commonly was found in mid and apical (66.67%) type of HCM followed by apical (16.7%) and reverse curvature (167%) type.

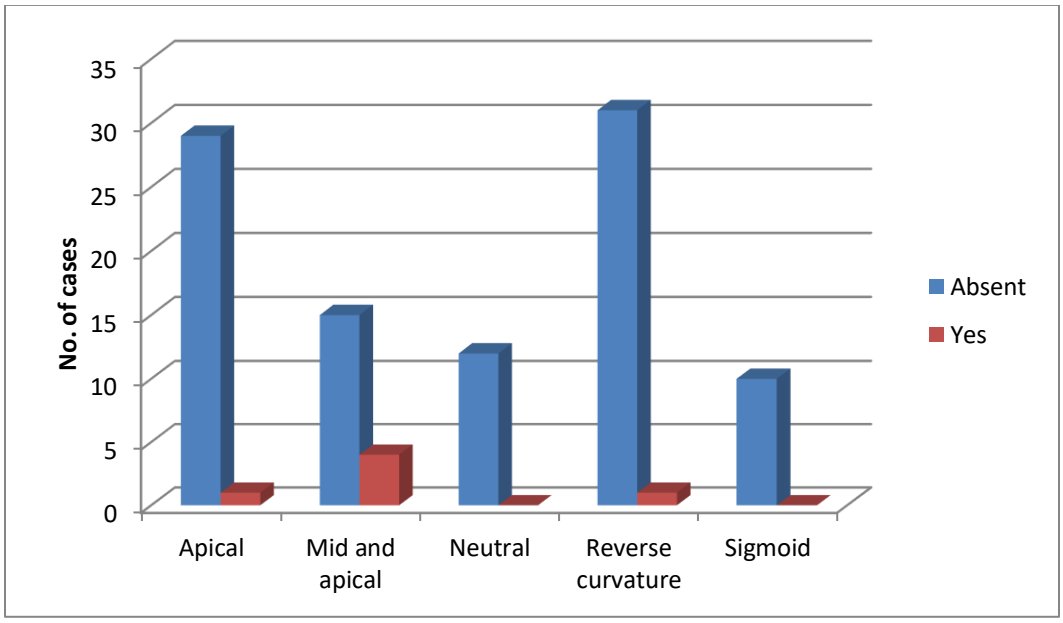

Figure 17: Comparison of mid-cavity gradient in various types of HCM patients

Table 20: Comparison of genetic analysis with types of HCM patients

	Genetic analysis(if required)									Total	Chi-square value	p- value
Type	CALR3	DTNA	MYBPC3	MYH6	MYH7	No abnormal gene found	Not done	PSEN2	TMEM43			
Apical	0	0	4	0	1	6	19	0	0	30	35.042	0.326
Mid and apical	0	0	2	0	1	7	9	0	0	19		
Neutral	0	0	1	0	1	2	7	1	0	12		
Reverse curvature	1	1	2	0	6	7	14	0	1	32		
Sigmoid	0	0	1	1	2	0	6	0	0	10		
Total	1	1	10	1	11	22	55	1	1	103		

MYH7 gene mutation was most commonly found in reverse curvature (54.5% i.e, 6 out of total 11 gene positive) type, where as MYBPC3 gene mutation was most commonly found in apical type (40% i.e, 4 out of total 10 gene positive) of HCM.

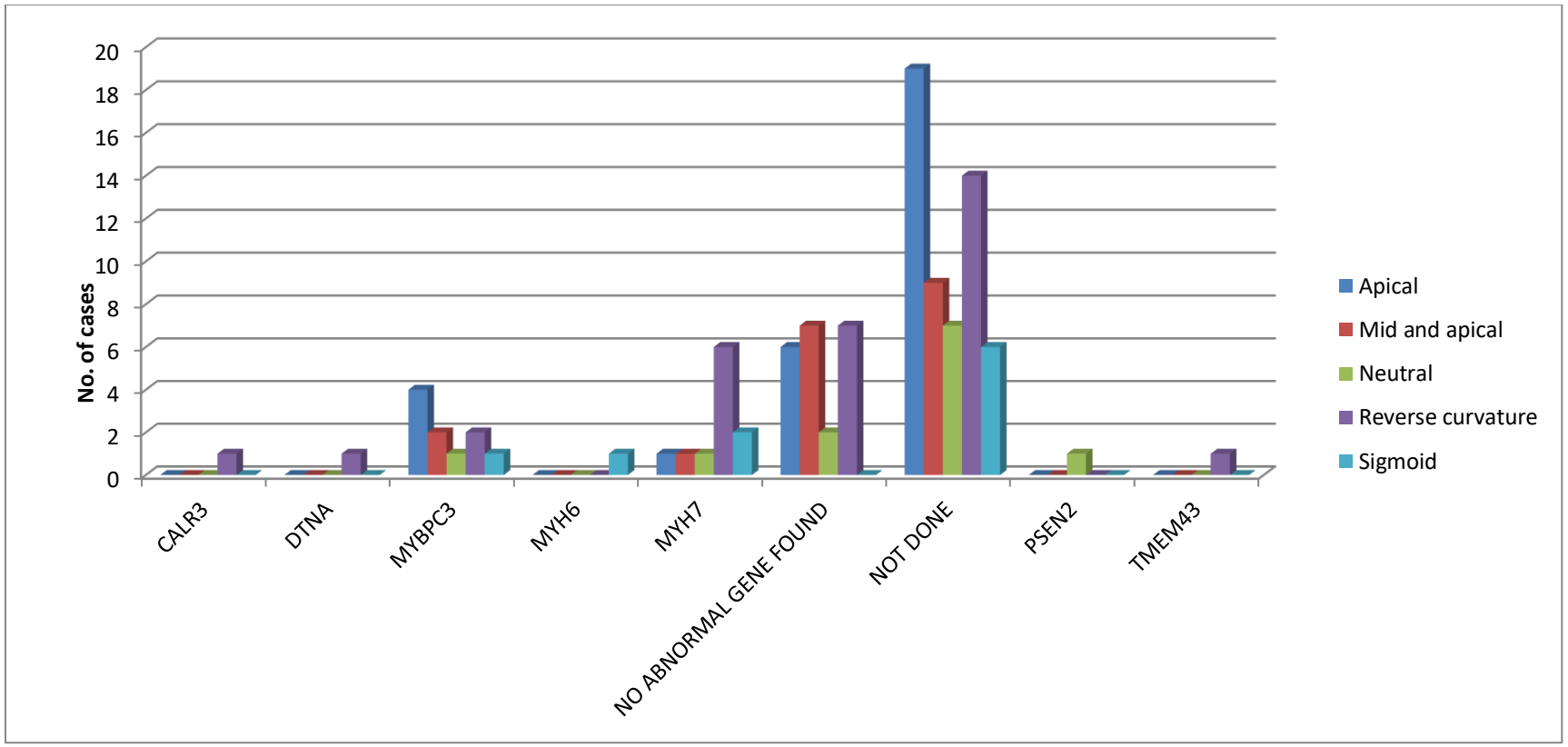

Figure 18: Comparison of genetic analysis with types of HCM patients

Table 21: Gender wise distribution of HCM patients according to the genes identified

		Sex				Total	Chi-square value	p-value
		F		M				
Genetic analysis (if required)	CALR3	0	0.0%	1	3.0%	1	0.464	0.496
	DTNA	1	6.7%	0	0.0%	1	2.247	0.134
	MYBPC3	3	20.0%	7	21.2%	10	0.009	0.924
	MYH6	0	0.0%	1	3.0%	1	0.464	0.496
	MYH7	6	40.0%	5	15.2%	11	3.605	0.074
	No abnormal gene found	5	33.3%	17	51.5%	22	1.373	0.351
	PSEN2	0	0.0%	1	3.0%	1	0.464	0.496
	TMEM43	0	0.0%	1	3.0%	1	0.464	0.496
Total		15	100.0%	33	100.0%	48		

Genetic analysis was done in a total of 48 (46.6%) out of 103 patients, 33 (68.8%) of which were were male and 15 (31.3%) were female. No abnormal gene was found in total of 22 (45.8%) out of 48 patients.

Most common gene found was MYH7 in a total 11 (22.19%) patients i.e, in 15.2% M and 40% F.

Second most common gene was found to be MYBPC3 in a total of 10 (20.83%) patients i.e, in 21.2% M and 20% F.

Less common genes were CALR3, DTNA, MYH6, PSEN2, and TMEM43.

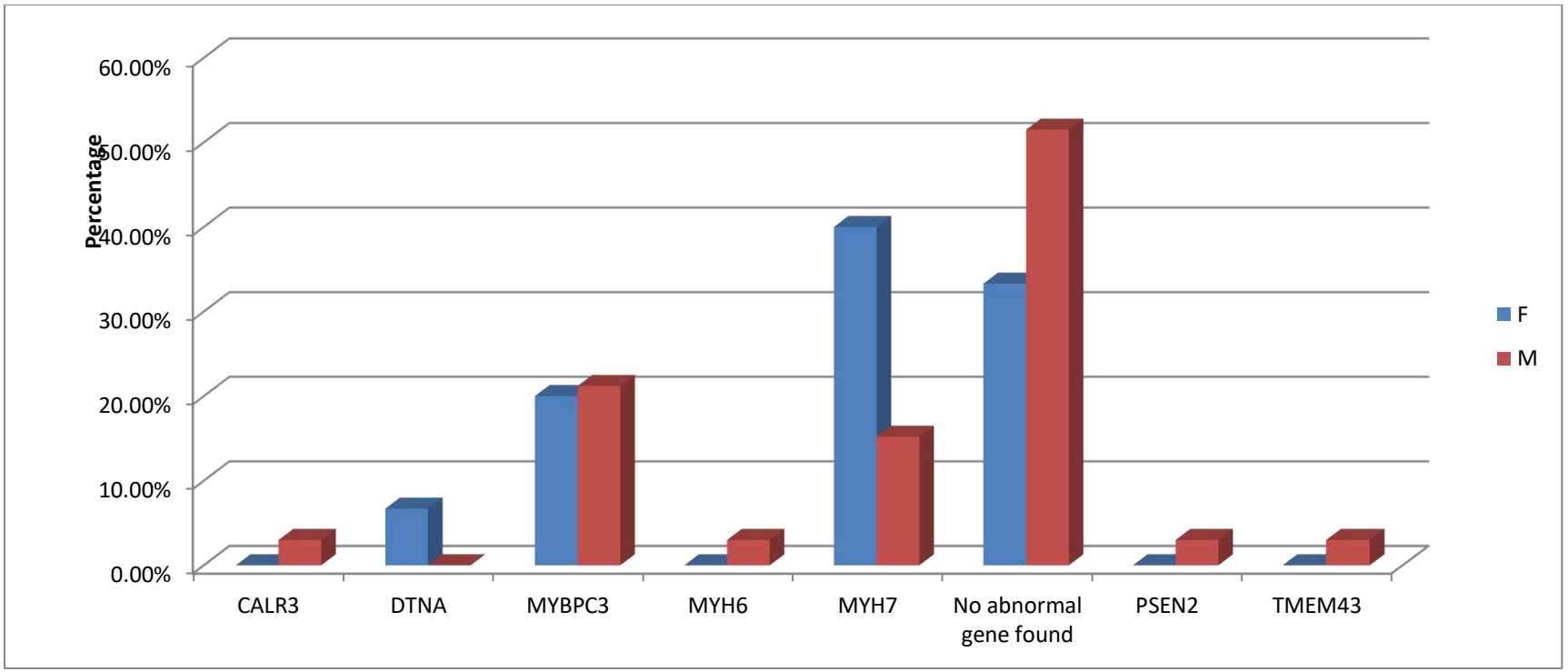

Figure 19: Gender wise distribution of HCM patients according to the genes identified

Table 22: Comparison of genetic analysis with family history of SCD

Genetic analysis	Family History of SCD		Total
	No	Yes	
CALR3	1	0	1
DTNA	1	0	1
MYBPC3	3	7	10
MYH6	0	1	1
MYH7	3	8	11
NO ABNORMAL GENE FOUND	15	7	22
NOT DONE	55	0	55
PSEN2	1	0	1
TMEM43	1	0	1
Total	80	23	103

Out of total 103 patients, family history of sudden cardiac death was found in total 23 (22.3%) out of 103 patients.

MYH7 gene was found in 34.8% (8 out of 23) of patients who had family history of SCD.

MYBPC3 gene was found in 30.4% (7 out of 23) of patients who had family history of SCD.

Other less common gene found was MYH6 in 4.3% (1 out of 23) and no abnormal gene was found in 30.4% (7out of 23) of patients.

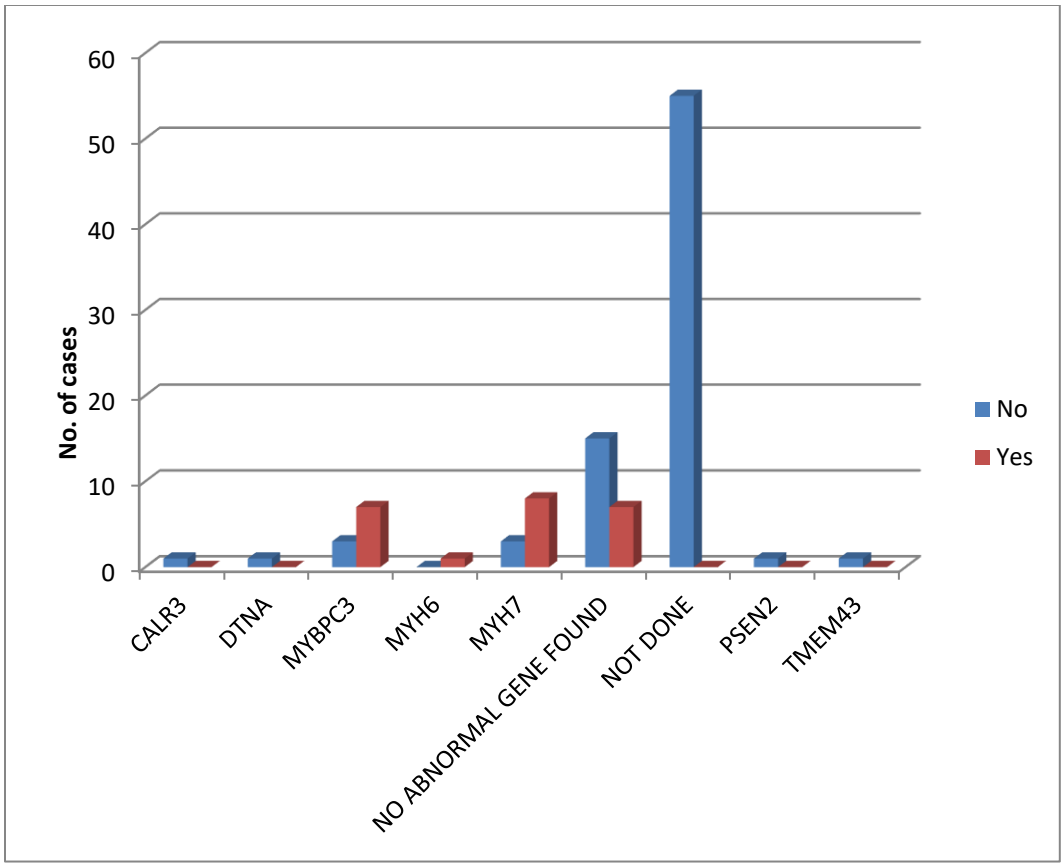

Figure 20: Comparison of genetic analysis with family history of SCD

Table 23: Comparison of significant LVOT gradient at rest/valsalva with clinical manifestation of presyncope/syncope.

		Presyncope/Syncope		Total	Chi-square value	p-value
		No	Yes			
LVOT gradient (≥30mm of Hg)	No	64	11	75	1.51	0.219
	Yes	21	7	28		
Total		85	18	103		

Presyncope/syncope was present in 7 (25%) out of total 28 patients when significant LVOT gradient at rest/valsalva was present (≥30mm of Hg) and was absent in 64 (85.3%) out of total 75 patients when significant LVOT gradient at rest/valsalva was absent (<30mm of Hg).

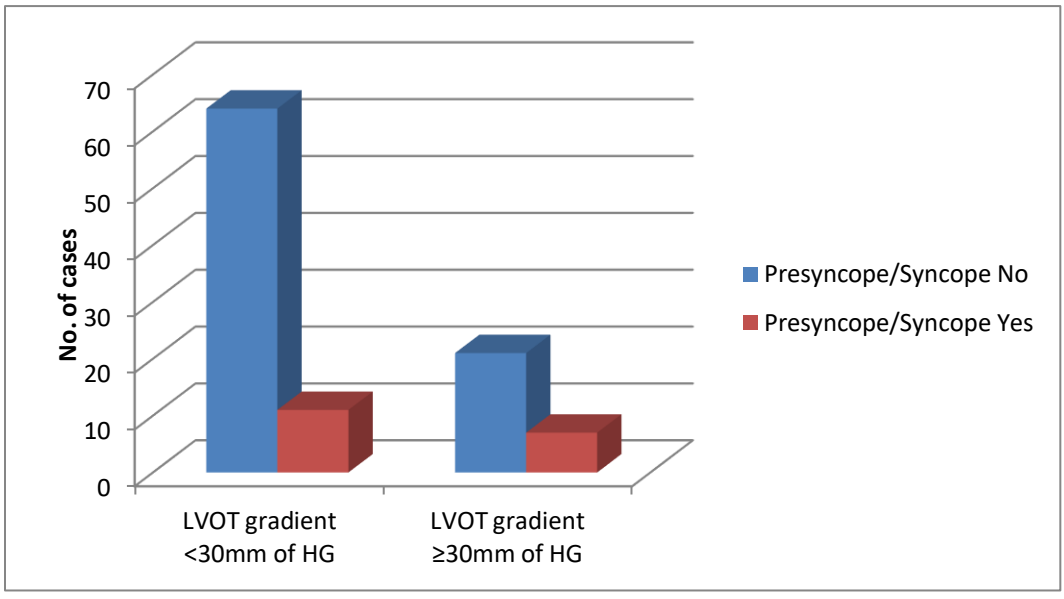

Figure 21: Comparison of significant LVOT gradient at rest/valsalva with clinical manifestation of presyncope/syncope

Table 24: Comparison of significant LVOT gradient at rest/valsalva with auscultation of ejection systolic murmur

		Auscultation ESM – Rest/valsalva			Chi-square value	p-value
		No	Yes	Total		
LVOT gradient (≥30mm of Hg)	No	67	8	75	45.542	0.0001
	Yes	6	22	28		
Total		73	30	103		

Ejection systolic murmur at rest/valsalva was present in 22 (78.6%) out of 28 patients when significant LVOT gradient at rest/valsalva was present (≥30mm of Hg) and murmur was absent in 67 (89.3%) out of 75 patients when significant LVOT gradient at rest/valsalva was absent (<30mm of Hg).

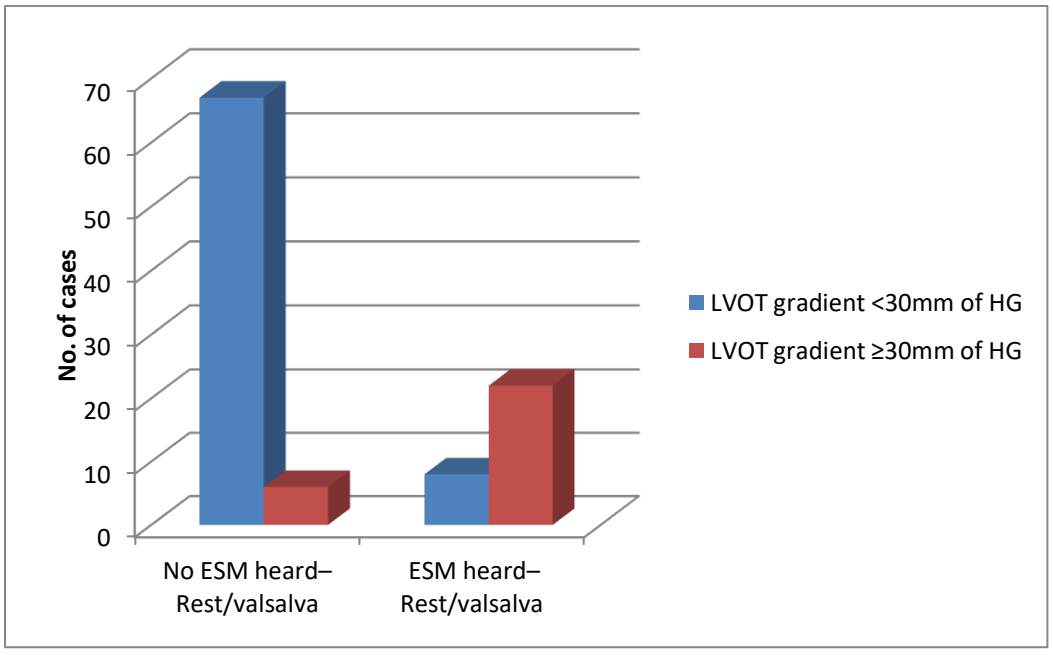

Figure 22: Comparison of significant LVOT gradient at rest/valsalva with auscultation of ejection systolic murmur

Table 25: Number of HCM patients with LVOT gradient at rest

LVOT gradient: resting (mm of Hg)	Frequency	Percent
< 30	4	14.82%
≥ 30	23	85.18%
Total	27	100%

Out of total 27 (26.2%) patients with resting LVOT gradient, 4(14.8%) patients had <30mm of Hg and 23(85.2%) patients had ≥30mm of Hg LVOT gradient.

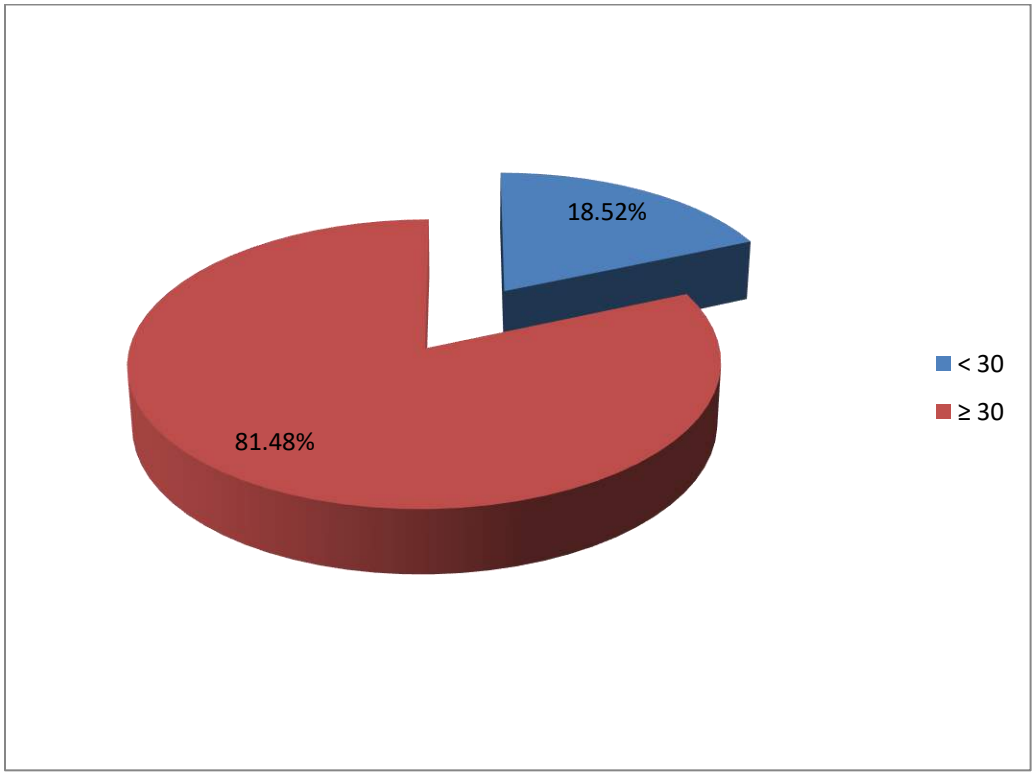

Figure 23: Number of HCM patients with LVOT gradient at rest

Table 26: Number of HCM patients with LVOT gradient at valsalva

LVOT gradient: valsalva(mm of Hg)	Frequency	Percent
<30	3	33.3%
≥30	6	66.67%
Total	9	100%

Out of total 9 (8.7%) patients with LVOT gradient at valsalva, 3 (33.3%) patients had <30mm of Hg and 6(66.7%) patients had ≥30mm of Hg LVOT gradient.

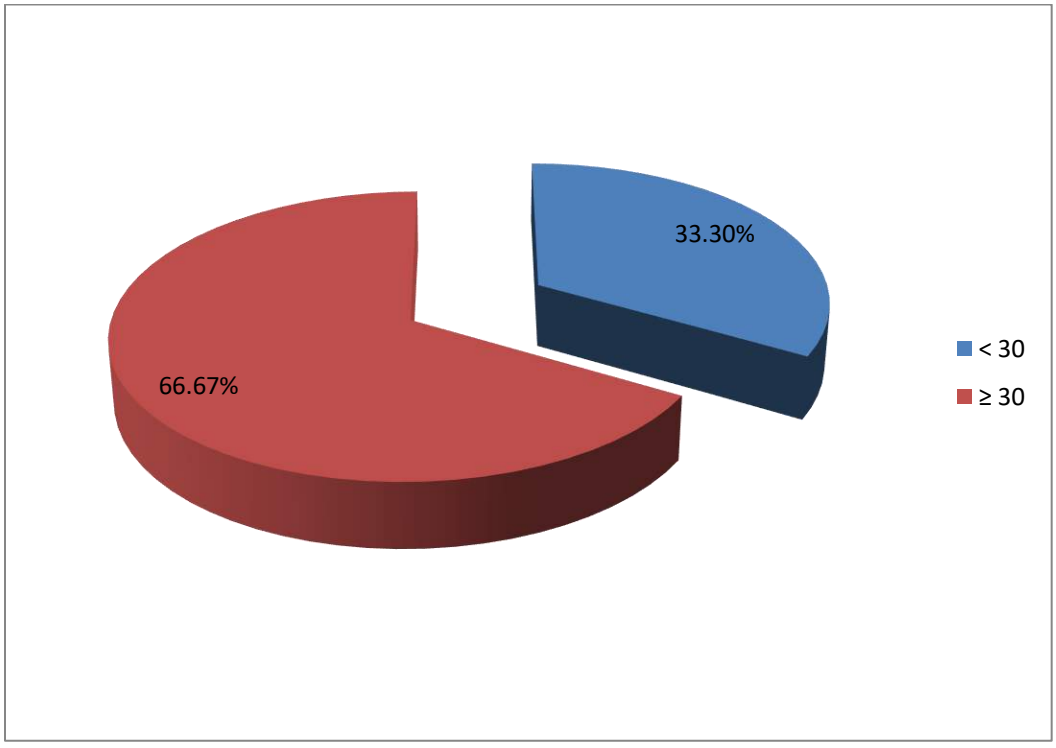

Figure 24: Number of HCM patients with LVOT gradient at valsalva

DISCUSSION

Table 27: Comparison of present study with previous studies in HCM patients

Studies	M:F prevalence	Mean age (years)	Past history of HTN	Genetic mutation	Obstructive v/s nonobstructive %	2D ECHO features		Variants of HCM with gender distribution and prevalence					Most common clinical presentation
						Mean posterior wall thickness (mm)	Mean IVS thickness (mm)	Reverse curvature	Apical	Mid and apical	Neutral	Sigmoid	
Tanjore R R et al [25](2006)	3.7:1	-	-	-	-	-	-	-	-	-	-	-	-
Rangaraju A et al[26] (2014)	3.1:1	38±10	-	-	-	-	-	-	-	-	-	-	-
Biswas A et al[27] (2015)	5.5:1	39±14.5	-	-	33.9% v/s 66.1%	16±4.8	21±8.3	-	-	-	-	-	-
Marian AJ et al[28] (2017)	-	-	-	MYH7 and MYBPC3 (60%)	-	-	-	-	-	-	-	-	-
Erica Zaiser et al[32] (2020)	-	-	-	-	-	-	-	-	-	-	-	-	Shortness of breath (89%)
Luo Q et al[29] (2020)	-	-	34.3%	-	-	-	-	-	-	-	-	-	-
Tandon Ret al[30] (2020)	2.3:1	53±14.5	-	-	-	-	-	M:F 3:1	M:F 2:1	M:F 2.4:1	M:F 2:1	M:F 2.5:1	
	-	-	-	-	-	-	-	49%	-	19%	11%	6% (Atypical 4%)	-
Inki Moon et al[31] (2020)	2:1	63±13.2	-	-	-	-	-	-	-	-	-	-	Chest pain (44.2%)
Luis R. Lopes et al[33](2021)	5.5:1	-	-	-	-	-	-	-	-	-	-	-	-
Present study	2.12:1	56±13.9	57%	MYH7 and MYBPC3 (44%)	25% v/s 75%	13.79±3.18	21.83±6.56	M:F 0.5:1	M:F 1.88:1	M:F 1.3:1	M:F 5.2:1	M:F 0.5:1	Shortness of breath (77.67%)
								31%	29%	18%	12%	10%	

The present study shows a male predominance over female with ratio of 2.12:1 which is concordance with the previous studies Tanjore RR et al[25] (2006), Rangaraju A et al[26] (2014), Biswas A et al[27] (2015), Tandon Ret al[30] (2020), Inki Moon et al[31] (2020), Luis R. Lopes et al[33] (2021).

The present study shows a mean age of onset in HCM patients during sixth and seventh decade which is concordance with the previous study of Tandon Ret al[30] (2020) and Inki Moon et al[31] (2020) where as other studies by Rangaraju A et al[26] (2014) and Biswas A et al[27] (2015) shows mean age of onset during fourth decade.

The present study shows past history of HTN in total 57% of patients where as in previous study by Luo Q et al[29] (2020) it was 34.3%.

The present study shows predominant gene mutation is MYH7 and MYBPC3 and is found in 44% of HCM patients which is in concordance with previous study by Marian A Jet al[28] (2017).

The present study shows predominance of nonobstructive (74.76%) type of HCM than obstructive (25.24%) which is in concordance with previous study by Biswas A et al[27] (2015).

The present study shows mean posterior wall and IVS thickness of 13.79±3.18 and 21.83±6.56mm which is in concordance with previous study of Biswas A et al[27] (2015).

The present study shows most prevalent type is reverse curvature (31%) and least prevalent is sigmoid (10%) which is in concordance with the previous study by Tandon R et al[30] (2020).

The present study shows M:F distribution is higher among types in apical, mid and apical, neutral variety while it is lower in reverse curvature and sigmoid type.

The present study shows most common clinical presentation was shortness of breath in 77.67% of patients which is in concordance with the previous study by Erica Zaiser et al[32] (2020) where as the study by Inki Moon et al[31] (2020) shows that most common clinical presentation was chest pain.

SUMMARY AND CONCLUSIONS

1. HCM was predominant in male sex with sex ratio of 2.1:1 (M:F).
2. Shortness of breath was the most common presentation (77.67%).
3. 71.84% of patients revealed past history of HTN.
4. Giant T wave inversion and left ventricular hypertrophy on ECG were most commonly found in apical type of HCM (46% and 38% respectively).
5. Nonobstructive type of HCM was more common than obstructive type (75% v/s 25%).
6. Reverse curvature was the commonest echocardiographic variant noted (31.1%).
7. The ventricular cavity dimensions were significantly smaller in female patients (LVID-S and D were 26.12±5.17mm and 41.09±6.09mm).
8. Mean maximum wall thickness and opposing wall ratio were 24.71±3.99mm and 2.34±0.70 respectively.
9. SAM with mitral regurgitation was typically found in reverse curvature type of HCM (79.17%).
10. MYH7 and MYBPC3 were the commonest genetic mutations noted (22.19% and 20.8% respectively).
11. Family history of sudden cardiac death was usually noted in the patients with genetic mutation for MYH7 (34.8%) and MYBPC3 (30.4%).

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Sr. No	MRD No.	Age	Sex	Chief Complaints					Diagnosis	Type	Past History							Personal History				Family History	Examination				Systemic Examination		
				Chest pain	SOB	Palpitations	Syncope	Presyncope			HTN	Bronchial Asthma	Arrythmia	CVA	DM	Heart failure	AICD implantation	Smoker/ Non Smoker	Alcoholic/ Non-Alcoholic	OSA	Marathon runner	SCD	Oedema	Pulse (/min)	SBP (mm Hg)	DBP (mm Hg)	Auscultation ESM – Rest/ Dynamic	Axis (degree)	Axis group
1	1130829	63	M	NO	NO	YES	YES	NO	HCMP	REVERSE CURVATURE	NO	NO	YES=AF	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	80	110	80	YES	-45	Left
2	1124735	60	F	YES	YES	NO	NO	NO	HCMP WITH ACS	REVERSE CURVATURE	YES	NO	NO	NO	YES	YES	NO	NON	NON	YES	NO	YES	YES	85	120	70	YES	-20	Normal
3	154171	67	F	NO	YES	NO	YES	NO	HCMP	SIGMOID	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	YES	YES	82	106	70	YES	-60	Left
4	937458	48	M	NO	YES	NO	NO	NO	HCMP	MID AND APICAL	YES	NO	NO	NO	NO	NO	NO	NON	YES	YES	NO	NO	NO	78	150	90	NO	0	Normal
5	939183	56	F	YES	NO	NO	NO	NO	HCMP WITH ACS	REVERSE CURVATURE	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	100	150	90	NO	0	Normal
6	268447	53	M	NO	NO	YES	NO	NO	HCMP	MID AND APICAL	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	62	120	70	YES	10	Normal
7	994341	60	M	NO	YES	NO	NO	NO	HCMP	MID AND APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	80	140	80	NO	10	Normal
8	972710	40	M	YES	NO	NO	NO	NO	HCMP	APICAL	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	YES	NO	78	120	70	NO	45	Normal
9	964838	68	M	NO	YES	NO	NO	NO	HCMP	SIGMOID	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	80	140	90	NO	-10	Normal
10	975260	54	F	NO	YES	NO	YES	NO	HCMP	MID AND APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	74	140	80	YES	-45	Left
11	1112572	41	M	NO	NO	NO	NO	YES	HCMP	NEUTRAL	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	78	130	80	NO	-20	Normal
12	949716	85	F	YES	YES	YES	YES	NO	HCMP	REVERSE CURVATURE	YES	NO	NO	NO	NO	NO	NO	NON	NON	YES	NO	NO	NO	82	110	70	YES	-30	Normal
13	1111934	58	M	NO	YES	NO	NO	NO	HCMP	APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	74	130	90	NO	60	Normal
14	947888	51	M	YES	YES	NO	NO	NO	HCMP	MID AND APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	72	130	90	YES	-30	Normal
15	938464	43	M	NO	YES	NO	NO	NO	HCMP	MID AND APICAL	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	74	120	70	NO	30	Normal
16	153895	39	F	NO	YES	NO	YES	NO	HCMP WITH HfPEF	SIGMOID	NO	NO	NO	NO	NO	YES	NO	NON	NON	NO	NO	NO	YES	50	100	60	NO	-45	Left
17	970037	50	F	NO	YES	NO	NO	NO	HOCM	REVERSE CURVATURE	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	82	150	90	YES	0	Normal
18	202198	58	F	NO	NO	NO	YES	NO	HCMP	APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	50	110	70	NO	30	Normal
19	924496	83	F	YES	NO	NO	NO	NO	HCMP WITH ACS	SIGMOID	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	60	140	80	NO	-30	Normal
20	666139	64	F	NO	YES	NO	NO	NO	HCMP	SIGMOID	YES	NO	NO	NO	NO	NO	NO	NON	NON	YES	NO	NO	YES	102	88	64	NO	0	Normal
21	812594	34	M	NO	NO	YES	NO	NO	HCMP	REVERSE CURVATURE	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	YES	NO	90	90	60	YES	-50	Left
22	162767	42	M	NO	YES	NO	NO	YES	HCMP	REVERSE CURVATURE	YES	NO	NO	NO	NO	NO	NO	NON	NON	YES	NO	NO	NO	68	140	90	YES	-20	Normal
23	1022391	68	F	NO	YES	NO	NO	NO	HCMP	APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	70	130	80	NO	45	Normal
24	1110582	50	M	NO	YES	NO	YES	NO	HCMP	REVERSE CURVATURE	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	79	140	90	YES	-10	Normal
25	1062310	34	M	NO	NO	NO	NO	NO	HCMP	APICAL	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	72	120	80	NO	0	Normal
26	1011983	52	M	NO	YES	NO	NO	NO	HCMP	MID AND APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	100	180	100	NO	-30	Normal
27	985393	66	M	NO	NO	NO	YES	NO	HCMP	MID AND APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	YES	NO	YES	NO	83	170	100	NO	-10	Normal

Sr. No	MRD No.	12 lead ECG						Echocardiographic Parameters																
		Rhythm	PR interval (ms)	QRS duration (ms)	LVH	QT_c (Bazett's) (ms)	Giant T wave inversion	Maximum wall thickness (mm)	Opposing wall ratio	LVID-S (mm)	LVID-D (mm)	LVPW-S (mm)	LVPW-D (mm)	IVS-S (mm)	IVS-D (mm)	LVOT gradient: resting (mm of Hg)	LVOT gradient: valsalva (mm of Hg)	MR	SAM	Mitral valve-E/A	E/e'	IVRT (ms)	DT (ms)	PASP (mm of Hg)	LAVI (ml/m2)
1	1130829	NSR	180	120	-	433	NO	31	2.80	33	49	12	11	40	32	46	-	YES, MILD	YES	-	18	84	166	40	38
2	1124735	NSR	160	120	-	467	NO	17	1.80	17	30	10	9	18	17	30	-	YES, MILD	YES	1.1	25	74	208	55	36
3	154171	NSR	160	120	YES	440	NO	22	3.38	22	45	7.1	6.5	22	20.3	-	24	NO	-	1	39	42	236	48	42
4	937458	NSR	160	120	YES	510	YES	28	1.64	27	47	21	17	24	20	-	-	NO	-	0.6	11.3	88	194	15	35
5	939183	NSR	160	120	-	520	YES	23	2.30	29	42	14	10	23	19	-	-	NO	-	0.6	19	96	165	32	36
6	268447	NSR	120	110	YES	440	YES	29	2.00	34	47	17	14	14	13	-	-	NO	-	0.5	10	113	222	20	33
7	994341	NSR	200	120	YES	472	YES	20	2.50	30	44	10	8	21	19	-	-	NO	-	2.1	14	80	172	25	36
8	972710	NSR	200	120	-	472	NO	25	-	37	49	13	11	25	23	-	30	NO	-	1.4	18	82	184	25	31
9	964838	NSR	180	120	-	463	NO	19	1.58	27	44	14	12	13	11	-	25	NO	-	0.6	12	74	162	35	36
10	975260	NSR	200	120	YES	483	YES	30	3.33	26	39	11	9	32	31	40	-	YES	YES	1	18	78	182	35	36
11	1112572	IRREGULAR	-	120	YES	493	NO	25	2.27	32	47	13	11	25	22	-	-	NO	-	1	12	70	202	30	36
12	949716	NSR	180	110	YES	452	NO	24	2.40	37	50	11	10	20	18	60	-	NO	YES	0.9	12	78	184	45	32
13	1111934	NSR	160	120	YES	462	YES	28	1.75	27	39	17	16	14	13	-	-	NO	-	1	12	76	192	20	36
14	947888	NSR	180	120	YES	493	NO	22	-	35	52	17	13	16	13	-	-	NO	-	0.5	12	78	177	40	36
15	938464	NSR	160	120	-	454	NO	29	-	33	50	17	13	16	13	-	-	NO	-	0.9	8	70	168	28	31
16	153895	PACEMAKER	200	200	-	440	NO	23	2.30	30	41	11	10	23	22	-	-	YES	-	1.5	16	98	156	40	45
17	970037	NSR	120	120	YES	467	YES	29	1.31	24	40	24	22	29	27	102	-	YES	-	0.5	19	69	198	25	34
18	202198	NSR	160	100	-	474	YES	22	-	32	48	16	14	16	14	-	40	YES	YES	1.9	15	89	158	40	32
19	924496	NSR	160	120	YES	453	NO	22	1.29	22	38	20	17	22	19	56	-	NO	YES	1.5	19	95	145	40	37
20	666139	IRREGULAR	-	120	-	444	NO	24	2.18	16	27	12	11	24	22	25	-	YES	YES	-	16	75	160	44	36
21	812594	NSR	160	100	YES	550	NO	26	1.23	17	24	23	21	26	22	58	-	YES, MODERATE	YES	2.8	23.8	63	190	54	50
22	162767	NSR	160	120	-	468	NO	28	2.00	26	40	17	14	31	28	28	45	NO	YES	0.6	18	90	180	35	28
23	1022391	NSR	120	80	YES	431	YES	28	-	24	50	10	8	10	8	-	-	YES	-	1.4	28	88	202	40	36
24	1110582	NSR	160	120	YES	483	NO	21	2.00	20	32	12	10	21	19	90	-	NO	YES	0.9	24	70	160	25	28
25	1062310	NSR	120	110	-	470	NO	24	-	24	29	10	8	13	12	-	-	NO	-	0.9	10	60	156	28	28
26	1011983	NSR	160	120	YES	464	YES	26	-	36	41	13	12	12	10	-	-	NO	-	1	17	75	161	24	33
27	985393	NSR	180	100	YES	445	YES	24	1.84	33	48	14	13	16	13	-	-	NO	-	1	20	60	123	30	38

Sr. No	MRD No.											24 hours Holter study (if required)	Genetic analysis (if required)	Radiological investigations	Cardiac MRI			
		LAEF= (MaxV-MinV) /MaxV%	EF by 2D echo (%)	EF by 3D echo (%)	Strain derived EF (%)	Stroke volume (ml)	LV MPI	GLS (-%)	GCS (-%)	LA strain (peak) %	Mid-cavity gradient				Ventricular thickness	SAM	LVOT obstruction	Variants of HCM
1	1130829	52	60	60	48	38	0.58	13	16	20	ABSENT	NOT DONE	NO ABNORMAL GENE FOUND	NOT DONE	-	-	-	-
2	1124735	48	35	38	32	42	0.54	18	19	21	ABSENT	NOT DONE	MYH7	NOT DONE	-	-	-	-
3	154171	44	60	60	51	56	0.64	11.2	16.5	19.5	ABSENT	NOT DONE	MYBPC3	NOT DONE	-	-	-	-
4	937458	47	60	60	49	31	0.77	4.5	4.2	17.7	YES	NOT DONE	NO ABNORMAL GENE FOUND	NOT DONE	-	-	-	-
5	939183	48	35	35	32	40	0.52	12	15.5	20.5	ABSENT	NOT DONE	MYH7	NOT DONE	-	-	-	-
6	268447	68	60	60	52	45	0.38	9	11.4	21	ABSENT	NOT DONE	NO ABNORMAL GENE FOUND	YES	20mm OF MID SEPTUM	NO	NO	-
7	994341	48	60	60	51	51	0.54	14	18	19	ABSENT	NOT DONE	NO ABNORMAL GENE FOUND	NOT DONE	-	-	-	-
8	972710	47.2	60	60	53	41	0.42	18	21.2	22.4	ABSENT	NOT DONE	MYBPC3	NOT DONE	-	-	-	-
9	964838	51	60	60	53	40	0.39	16	17.2	19	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
10	975260	56	60	60	51	43	0.61	13	15	18	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
11	1112572	51.3	60	60	52	42	0.59	15.2	18.4	23	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
12	949716	49	60	60	51	44	0.41	11	12.1	16	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
13	1111934	52	60	60	52	54	0.32	18.3	23.2	21.4	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
14	947888	51	60	60	42	42	0.51	13.1	18.1	21.2	YES	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
15	938464	51	60	60	52.3	51	0.44	17.2	22.1	21	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
16	153895	32	60	60	53	45	0.53	15.6	16.8	18.6	ABSENT	NOT DONE	MYH7	NOT DONE	-	-	-	-
17	970037	54	60	60	52	40	0.62	7	9	15	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
18	202198	56	60	60	50	59	0.45	16.5	19.8	22	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
19	924496	49	45	45	34	56	0.56	13.2	15.8	19	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
20	666139	58	60	60	55	60	0.51	16.2	14.2	23	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
21	812594	61	60	60	58	64	0.39	10.2	6.8	19.5	ABSENT	NOT DONE	MYH7	NOT DONE	-	-	-	-
22	162767	69	60	60	51	42	0.49	11.2	20.2	22	ABSENT	YES=NORMAL	TMEM43	NOT DONE	-	-	-	-
23	1022391	58	60	60	57	44	0.55	16	11.4	22.4	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
24	1110582	56.4	60	60	51	48	0.51	13.2	21.4	23	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
25	1062310	53	60	60	54	54	0.49	15.6	16.2	20.5	ABSENT	NOT DONE	MYBPC3	NOT DONE	-	-	-	-
26	1011983	45.5	60	60	58	60	0.49	9	10	20.8	YES	NOT DONE	NO ABNORMAL GENE FOUND	NOT DONE	-	-	-	-
27	985393	54	60	60	55	60	0.43	8.4	11.2	21.2	ABSENT	NOT DONE	MYH7	NOT DONE	-	-	-	-

Sr. No	MRD No.	Age	Sex	Chief Complaints					Diagnosis	Type	Past History							Personal History				Family History	Examination				Systemic Examination		
				Chest pain	SOB	Palpitations	Syncope	Presyncope			HTN	Bronchial Asthma	Arrythmia	CVA	DM	Heart failure	AICD implantation	Smoker/ Non Smoker	Alcoholic/ Non-Alcoholic	OSA	Marathon runner	SCD	Oedema	Pulse (/min)	SBP (mm Hg)	DBP (mm Hg)	Auscultation ESM – Rest/ Dynamic	Axis (degree)	Axis group
28	999475	60	M	NO	NO	YES	NO	NO	HCMP	NEUTRAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	YES	NO	70	140	90	NO	10	Normal
29	960191	44	M	NO	NO	YES	NO	YES	HCMP	NEUTRAL	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	63	130	80	NO	-10	Normal
30	1017004	40	M	YES	NO	NO	NO	NO	HCMP	NEUTRAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	YES	NO	88	140	90	NO	0	Normal
31	376870	74	M	NO	YES	NO	NO	NO	HCMP	MID AND APICAL	NO	NO	YES=AF	YES	NO	NO	NO	NON	NON	NO	NO	NO	NO	138	100	70	NO	30	Normal
32	169341	67	F	NO	YES	NO	NO	NO	HCMP WITH CAD	MID AND APICAL	YES	NO	NO	NO	YES	NO	NO	NON	NON	NO	NO	NO	YES	60	120	70	NO	80	Normal
33	937642	68	M	NO	NO	NO	YES	NO	HCMP WITH CKD	APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	60	120	70	NO	60	Normal
34	738009	70	M	NO	YES	NO	NO	NO	HCMP	APICAL	YES	NO	YES=AF	NO	NO	NO	NO	NON	YES	NO	NO	NO	NO	60	124	82	NO	0	Normal
35	302004	63	M	NO	YES	NO	NO	NO	HCMP	REVERSE CURVATURE	YES	NO	NO	NO	NO	NO	NO	NON	NON	YES	NO	NO	NO	87	150	74	YES	60	Normal
36	250097	64	M	NO	YES	NO	YES	NO	HCMP	APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	64	130	70	NO	90	Normal
37	267515	28	F	NO	YES	NO	NO	NO	HCMP	REVERSE CURVATURE	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	75	150	90	YES	-20	Normal
38	970037	56	F	NO	YES	NO	NO	NO	HCMP	REVERSE CURVATURE	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	92	150	80	NO	30	Normal
39	956230	64	M	NO	YES	NO	NO	NO	HCMP	SIGMOID	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	YES	NO	50	144	92	NO	0	Normal
40	975248	70	M	YES	YES	NO	NO	NO	HCMP WITH CAD	APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	94	110	60	YES	30	Normal
41	970037	56	F	NO	YES	YES	NO	NO	HCMP	REVERSE CURVATURE	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	69	130	70	YES	20	Normal
42	920891	45	F	NO	NO	NO	NO	NO	HCMP WITH CVA	NEUTRAL	YES	NO	NO	YES	NO	NO	NO	NON	NON	NO	NO	YES	NO	101	160	80	NO	-10	Normal
43	1009048	73	F	NO	NO	NO	NO	YES	HCMP	MID AND APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	78	130	80	NO	-45	Left
44	991739	39	M	NO	NO	NO	NO	YES	HCMP	NEUTRAL	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	75	122	82	NO	-10	Normal
45	1011304	55	M	NO	YES	NO	NO	NO	HCMP	REVERSE CURVATURE	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	YES	89	140	80	NO	60	Normal
46	1012280	62	M	NO	NO	NO	NO	NO	HCMP	APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	YES	NO	NO	NO	108	140	90	NO	-30	Normal
47	1047867	62	F	YES	YES	NO	NO	NO	HCMP	REVERSE CURVATURE	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	74	140	90	YES	-30	Normal
48	386825	36	M	NO	YES	NO	NO	NO	HCMP	REVERSE CURVATURE	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	69	110	80	NO	-45	Left
49	200369	38	M	NO	YES	NO	NO	NO	HCMP	REVERSE CURVATURE	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	94	120	70	YES	-10	Normal
50	1018411	56	M	YES	NO	NO	NO	NO	HCMP	REVERSE CURVATURE	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	98	100	60	YES	20	Normal
51	1014117	31	M	NO	NO	NO	NO	NO	HCMP	APICAL	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	YES	NO	74	110	80	NO	-20	Normal
52	672843	62	M	NO	YES	NO	NO	NO	HCMP	APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	103	140	90	NO	-10	Normal
53	554400	37	M	NO	YES	NO	NO	NO	HCMP	NEUTRAL	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	78	120	70	NO	-10	Normal
54	563361	59	F	NO	YES	YES	NO	NO	HCMP	REVERSE CURVATURE	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	82	150	100	NO	-30	Normal

Sr. No	MRD No.	12 lead ECG						Echocardiographic Parameters																	
		Rhythm	PR interval (ms)	QRS duration (ms)	LVH	QT_c (Bazett's) (ms)	Giant T wave inversion	Maximum wall thickness (mm)	Opposing wall ratio	LVID-S (mm)	LVID-D (mm)	LVPW-S (mm)	LVPW-D (mm)	IVS-S (mm)	IVS-D (mm)	LVOT gradient: resting (mm of Hg)	LVOT gradient: valsalva (mm of Hg)	MR	SAM	Mitral valve-E/A	E/e'	IVRT (ms)	DT (ms)	PASP (mm of Hg)	LAVI (ml/m2)
28	999475	NSR	100	110	YES	390	YES	27	2.45	24	35	12	11	27	26	-	-	NO	-	1.6	13	70	168	25	32
29	960191	NSR	120	110	-	405	NO	24	2.40	30	34	11	10	24	21	-	-	NO	-	1.2	9	108	204	25	28
30	1017004	NSR	160	120	YES	431	YES	24	1.60	24	40	19	15	24	22	-	-	NO	-	1.1	12.7	61	98	25	31
31	376870	IRREGULAR	-	120	-	434	NO	26	3.25	26	43	11	8	26	23	-	-	YES	-	0.8	20	60	180	50	45
32	169341	PACEMAKER	120	160	-	480	YES	22	3.14	36	47	10	7	22	20	-	-	YES	-	1.6	18	96	189	45	39
33	937642	NSR	160	120	YES	440	YES	18	-	33	49	14	13	13	12	-	-	NO	-	1.6	12.2	77	205	40	37
34	738009	IRREGULAR	-	80	YES	540	YES	20	-	38	46	16	11	17	13	-	-	NO	-	-	11.8	88	232	20	48
35	302004	NSR	200	120	-	476	NO	22	2.20	32	48	11	10	22	20	98	-	YES, MODERATE	YES	1	25	78	194	35	32
36	250097	NSR	160	120	YES	505	YES	22	2.00	35	52	13	11	23	20	-	-	NO	-	1.5	22	80	166	30	38
37	267515	NSR	160	120	YES	450	YES	33	4.12	25	39	12	8	33	30	60	-	YES, MODERATE	YES	1.6	22	96	186	35	40
38	970037	NSR	120	110	YES	415	YES	29	2.90	24	40	13	10	29	27	80	-	YES, MODERATE	YES	2	19	53	178	25	36
39	956230	NSR	160	120	-	403	NO	23	1.27	20	34	21	18	23	15	-	-	NO	-	1.4	8	56	160	35	34
40	975248	NSR	120	110	YES	432	YES	15	-	37	51	14	12	15	13	-	-	NO	-	1.5	12	75	146	20	32
41	970037	NSR	120	120	-	430	YES	29	2.90	24	40	13	10	29	27	80	-	YES, MODERATE	YES	0.5	18	92	185	30	30
42	920891	NSR	160	120	YES	551	YES	33	4.12	24	36	11	8	33	28	-	-	NO	-	0.8	12	56	110	20	36
43	1009048	NSR	160	120	YES	433	YES	23	2.30	27	38	11	10	23	21	-	-	YES	-	0.7	13.4	49	130	15	32
44	991739	NSR	160	120	-	416	YES	23	1.91	17	32	14	12	25	22	-	-	NO	-	1.4	9	90	120	30	31
45	1011304	NSR	160	120	YES	476	YES	27	2.45	17	30	12	11	27	25	-	-	NO	-	2.1	15	46	158	55	40
46	1012280	NSR	160	120	YES	434	YES	28	2.80	37	52	11	10	12	11	-	-	NO	-	1	20	74	260	25	32
47	1047867	NSR	120	110	YES	487	YES	28	2.54	22	40	12	11	26	24	-	-	NO	-	0.5	11	113	222	20	32
48	386825	NSR	140	110	-	434	NO	24	2.40	36	54	11	10	26	24	-	-	NO	-	1.2	15	77	183	35	32
49	200369	NSR	120	110	-	430	NO	27	1.58	27	38	19	17	27	25	28	45	YES	YES	1.2	12	65	110	25	36
50	1018411	NSR	160	110	YES	506	YES	33	3.60	33	49	12	9	31	28	52	-	YES, MODERATE	YES	1.5	12	94	63	22	28
51	1014117	NSR	160	120	YES	414	YES	23	-	31	48	13	11	13	10	-	-	YES	-	1.3	9	46	203	30	38
52	672843	NSR	160	120	YES	412	YES	27	-	37	49	17	14	16	14	-	-	NO	-	2.4	12.1	63	161	50	35
53	554400	NSR	160	120	-	462	NO	25	3.12	26	41	11	8	23	22	-	-	NO	-	1	10.6	40	108	22	29
54	563361	NSR	160	120	-	475	YES	30	2.00	28	46	18	15	33	30	-	-	NO	-	0.7	14	84	166	25	32

Sr. No	MRD No.											24 hours Holter study (if required)	Genetic analysis (if required)	Radiological investigations	Cardiac MRI			
		LAEF= (MaxV-MinV) /MaxV%	EF by 2D echo (%)	EF by 3D echo (%)	Strain derived EF (%)	Stroke volume (ml)	LV MPI	GLS (-%)	GCS (-%)	LA strain (peak) %	Mid-cavity gradient				Ventricular thickness	SAM	LVOT obstruction	Variants of HCM
28	999475	59	60	60	59	65	0.39	19.4	18.6	22.9	ABSENT	YES=RUNS OF NSVT	MYBPC3	NOT DONE	-	-	-	-
29	960191	22	60	60	51	60	0.45	18.4	21	15	ABSENT	YES=NORMAL	NO ABNORMAL GENE FOUND	NOT DONE	-	-	-	-
30	1017004	26	60	60	56	52	0.53	7.2	10.5	20	ABSENT	NOT DONE	MYH7	NOT DONE	-	-	-	-
31	376870	59	60	60	58	63	0.51	11.2	9.3	15.2	ABSENT	NOT DONE	MYBPC3	NOT DONE	-	-	-	-
32	169341	49	46	46	35	40	0.69	12.3	10.9	12	ABSENT	NOT DONE	NO ABNORMAL GENE FOUND	NOT DONE	-	-	-	-
33	937642	61.7	60	65	55	97	0.52	17.2	26.1	26.6	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
34	738009	41	60	60	49	55	0.53	6.9	14.4	6.1	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
35	302004	60	60	60	54	80	0.38	5.4	4	37.8	ABSENT	NOT DONE	CALR3	NOT DONE	-	-	-	-
36	250097	58	60	60	55	80	0.65	14	20	27.9	ABSENT	NOT DONE	NO ABNORMAL GENE FOUND	NOT DONE	-	-	-	-
37	267515	45	60	60	52	52	0.59	19.2	20.5	20.2	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
38	970037	56	60	60	53	68	0.52	7	10	15	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
39	956230	53	60	60	58	60	0.45	12	8	21	ABSENT	NOT DONE	MYH6	NOT DONE	-	-	-	-
40	975248	58	60	60	51	55	0.42	15	18	22	YES	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
41	970037	52	60	60	53	60	0.5	7	9	15	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
42	920891	62	60	60	59	62	0.41	19.2	20	20	ABSENT	NOT DONE	NO ABNORMAL GENE FOUND	NOT DONE	-	-	-	-
43	1009048	60	60	60	58	55	0.48	20.1	23	21	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
44	991739	55	60	60	51	58	0.44	18.4	16.4	18	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
45	1011304	48	60	60	56	60	0.49	18.1	17.2	19	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
46	1012280	41	60	60	53	54	0.48	9	25	20	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
47	1047867	56	60	60	48	42	0.45	9.8	13.4	23	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
48	386825	62	60	60	51	55	0.46	13	17	23	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
49	200369	59	60	60	58	45	0.53	16.4	13.1	21	ABSENT	NOT DONE	NO ABNORMAL GENE FOUND	NOT DONE	-	-	-	-
50	1018411	68	60	60	53	50	0.56	9	6.4	17.3	ABSENT	NOT DONE	NO ABNORMAL GENE FOUND	NOT DONE	-	-	-	-
51	1014117	57	60	60	44	60	0.51	15.1	24.8	23	ABSENT	NOT DONE	NO ABNORMAL GENE FOUND	NOT DONE	-	-	-	-
52	672843	22	60	60	53	40	0.61	9.2	22.1	22	ABSENT	NOT DONE	NO ABNORMAL GENE FOUND	NOT DONE	-	-	-	-
53	554400	59	60	60	51	58	0.52	12.4	18.6	21	ABSENT	NOT DONE	PSEN2	YES	18	NO	-	-
54	563361	61	60	60	53	45	0.43	16.3	20.1	21.2	ABSENT	NOT DONE	DTNA	NOT DONE	-	-	-	-

Sr. No	MRD No.	Age	Sex	Chief Complaints					Diagnosis	Type	Past History							Personal History				Family History	Examination				Systemic Examination		
				Chest pain	SOB	Palpitations	Syncope	Presyncope			HTN	Bronchial Asthma	Arrythmia	CVA	DM	Heart failure	AICD implantation	Smoker/ Non Smoker	Alcoholic/ Non-Alcoholic	OSA	Marathon runner	SCD	Oedema	Pulse (/min)	SBP (mm Hg)	DBP (mm Hg)	Auscultation ESM – Rest/ Dynamic	Axis (degree)	Axis group
55	1026495	37	M	NO	YES	NO	NO	NO	HCMP	REVERSE CURVATURE	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	YES	NO	62	100	70	YES	-30	Normal
56	1108381	75	M	NO	YES	NO	NO	NO	HCMP	REVERSE CURVATURE	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	81	130	80	YES	-30	Normal
57	851649	66	F	NO	YES	YES	NO	NO	HCMP	REVERSE CURVATURE	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	72	130	80	YES	60	Normal
58	382547	63	M	NO	YES	NO	NO	NO	HCMP	APICAL	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	69	120	70	NO	30	Normal
59	1047867	62	F	NO	YES	NO	NO	NO	HCMP	REVERSE CURVATURE	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	YES	NO	84	140	80	NO	-20	Normal
60	386825	38	M	NO	YES	NO	NO	NO	HCMP	NEUTRAL	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	72	130	80	NO	30	Normal
61	1076244	53	M	YES	NO	NO	NO	NO	HCMP	NEUTRAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	79	140	90	NO	45	Normal
62	1082791	72	M	NO	YES	YES	NO	NO	HCMP	APICAL	YES	NO	NO	NO	NO	NO	NO	NON	YES	NO	NO	NO	NO	84	140	80	NO	-30	Normal
63	221596	66	M	NO	YES	NO	NO	NO	HCMP	APICAL	YES	NO	NO	NO	NO	NO	NO	NON	YES	NO	NO	YES	NO	80	140	80	NO	-10	Normal
64	1011493	70	M	NO	YES	NO	NO	NO	HCMP	REVERSE CURVATURE	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	YES	NO	84	130	80	NO	30	Normal
65	1083276	65	F	NO	YES	NO	NO	NO	HCMP	REVERSE CURVATURE	YES	NO	NO	NO	NO	NO	NO	NON	NON	YES	NO	NO	NO	80	130	80	YES	-20	Normal
66	1108381	64	M	NO	YES	NO	YES	NO	HCMP	REVERSE CURVATURE	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	82	130	90	YES	-30	Normal
67	1087012	47	F	NO	YES	YES	NO	NO	HCMP	REVERSE CURVATURE	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	68	140	80	YES	-10	Normal
68	1094704	49	M	YES	YES	NO	NO	NO	HCMP WITH CAD	SIGMOID	YES	NO	NO	NO	NO	NO	NO	YES	NON	NO	NO	NO	NO	78	130	80	NO	30	Normal
69	1052079	65	M	NO	YES	NO	NO	NO	HCMP	APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	82	140	90	NO	20	Normal
70	1053705	55	M	YES	YES	NO	NO	NO	HCMP WITH CAD	REVERSE CURVATURE	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	YES	NO	72	110	70	YES	-30	Normal
71	591665	90	M	NO	YES	NO	NO	NO	HCMP WITH CAD	SIGMOID	YES	NO	YES=AF	NO	NO	NO	NO	NON	NON	YES	NO	NO	NO	71	170	80	NO	10	Normal
72	34475	39	M	NO	YES	NO	NO	NO	HCMP	MID AND APICAL	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	82	120	70	YES	-10	Normal
73	41406	64	F	NO	NO	YES	NO	YES	HCMP	APICAL	YES	NO	YES=AF	NO	NO	NO	NO	NON	NON	NO	NO	YES	NO	50	110	70	YES	10	Normal
74	777590	62	M	YES	YES	YES	YES	NO	HCMP	SIGMOID	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	78	130	80	NO	20	Normal
75	875932	59	M	NO	YES	NO	NO	NO	HCMP	NEUTRAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	74	140	90	NO	-10	Normal
76	963403	39	M	NO	YES	NO	NO	NO	HCMP	APICAL	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	82	110	70	NO	-20	Normal
77	41406	64	F	NO	YES	NO	NO	NO	HCMP	APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	74	130	80	NO	0	Normal
78	1114348	59	F	NO	YES	YES	NO	NO	HCMP	REVERSE CURVATURE	YES	NO	YES=AF	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	95	130	80	NO	10	Normal
79	442020	41	M	NO	YES	NO	NO	NO	HCMP	REVERSE CURVATURE	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	82	140	90	NO	30	Normal
80	986034	60	F	NO	YES	YES	NO	NO	HCMP	MID AND APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	YES	NO	84	140	80	NO	30	Normal
81	991739	39	M	NO	YES	NO	NO	NO	HCMP	NEUTRAL	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	74	130	80	NO	30	Normal

Sr. No	MRD No.	12 lead ECG						Echocardiographic Parameters																
		Rhythm	PR interval (ms)	QRS duration (ms)	LVH	QT_c (Bazett's) (ms)	Giant T wave inversion	Maximum wall thickness (mm)	Opposing wall ratio	LVID-S (mm)	LVID-D (mm)	LVPW-S (mm)	LVPW-D (mm)	IVS-S (mm)	IVS-D (mm)	LVOT gradient: resting (mm of Hg)	LVOT gradient: valsalva (mm of Hg)	MR	SAM	Mitral valve-E/A	E/e'	IVRT (ms)	DT (ms)	PASP (mm of Hg)	LAVI (ml/m2)
55	1026495	NSR	160	120	YES	660	YES	27	1.92	35	49	17	14	16	14	21	44	YES, MODERATE	YES	0.7	9.4	88	236	30	31
56	1108381	NSR	120	110	YES	483	NO	20	1.53	18	23	18	13	24	21	105	-	YES, MODERATE	YES	0.9	24	77	296	35	38
57	851649	NSR	120	110	-	487	NO	38	4.22	22	40	13	9	37	30	56	-	YES, MODERATE	YES	0.7	14	56	384	40	33
58	382547	NSR	140	120	-	444	YES	30	-	27	48	19	15	16	14	-	-	NO	-	1	10	48	190	30	22
59	1047867	NSR	120	110	YES	474	NO	25	2.27	22	40	12	11	26	24	100	-	YES, MODERATE	YES	0.7	14	32	167	40	32
60	386825	NSR	160	120	-	434	NO	20	2.20	35	52	11	9	20	18	-	-	NO	-	0.8	10	74	190	25	19
61	1076244	NSR	160	120	-	498	NO	25	2.08	28	43	13	12	27	25	-	-	NO	-	0.7	10	74	109	30	21
62	1082791	NSR	160	120	YES	483	YES	25	-	31	49	16	14	18	14	-	-	YES	-	1.2	14	72	190	28	31
63	221596	NSR	200	120	YES	472	YES	21	1.75	30	45	13	12	14	12	-	-	YES	-	0.9	15	74	160	30	36
64	1011493	NSR	160	120	-	452	NO	20	2.20	33	49	12	9	20	18	-	-	NO	-	0.6	14	78	254	30	28
65	1083276	NSR	160	120	-	434	NO	18	1.80	32	49	12	10	19	18	54	-	YES	YES	0.9	12	79	189	25	30
66	1108381	NSR	160	120	-	484	NO	23	1.76	21	31	16	13	23	21	90	-	YES, MODERATE	YES	1.5	32	74	350	40	38
67	1087012	NSR	160	120	-	482	NO	28	2.15	23	38	19	13	28	25	20	42	YES	YES	1.8	15	84	289	20	36
68	1094704	NSR	160	120	-	484	NO	28	2.00	33	43	16	14	28	26	-	-	NO	-	0.9	15	74	263	30	33
69	1052079	NSR	120	110	YES	449	NO	21	-	27	39	17	16	14	13	-	-	NO	-	0.8	16	90	190	35	34
70	1053705	NSR	120	110	YES	430	YES	24	3.00	24	40	10	8	24	22	-	-	YES, MODERATE	YES	0.9	22	90	60	20	35
71	591665	IRREGULAR	-	120	-	432	NO	24	2.18	32	38	12	11	24	22	-	-	NO	-	-	10.6	97	183	25	38
72	34475	NSR	160	120	YES	466	YES	24	1.50	37	56	17	16	24	22	-	-	NO	-	1.6	19	66	201	30	28
73	41406	IRREGULAR	-	120	YES	435	YES	23	-	29	40	14	13	15	16	-	-	NO	-	1.9	18	70	155	30	32
74	777590	NSR	180	120	-	473	NO	24	2.66	34	44	10	9	20	18	-	26	YES	YES	0.6	18	78	198	20	34
75	875932	NSR	200	120	-	463	NO	28	3.11	41	56	10	9	28	26	-	-	NO	-	0.9	14	78	192	20	40
76	963403	NSR	120	110	YES	452	YES	20	-	33	53	14	10	13	10	-	-	NO	-	1.4	13	78	162	25	32
77	41406	NSR	180	120	-	463	NO	23	-	29	40	14	13	15	14	-	-	NO	-	1.6	19	79	182	30	38
78	1114348	IRREGULAR	-	110	-	452	NO	28	2.80	23	38	13	10	28	26	-	-	NO	-	-	20	78	144	50	40
79	442020	NSR	160	120	-	472	NO	28	2.30	50	59	14	12	28	27	-	-	NO	-	0.9	15	78	173	32	34
80	986034	NSR	160	120	-	474	NO	24	1.80	34	42	14	13	24	23	65	-	NO	-	2.2	28	92	156	25	36
81	991739	NSR	180	120	-	457	NO	23	2.30	17	32	11	10	23	21	-	-	NO	-	1.8	20	74	192	20	34

Sr. No	MRD No.											24 hours Holter study (if required)	Genetic analysis (if required)	Radiological investigations	Cardiac MRI			
		LAEF= (MaxV-MinV) /MaxV%	EF by 2D echo (%)	EF by 3D echo (%)	Strain derived EF (%)	Stroke volume (ml)	LV MPI	GLS (-%)	GCS (-%)	LA strain (peak) %	Mid-cavity gradient				Ventricular thickness	SAM	LVOT obstruction	Variants of HCM
55	1026495	43	60	60	52	30	0.52	10.4	13.8	31	ABSENT	NOT DONE	NO ABNORMAL GENE FOUND	NOT DONE	-	-	-	-
56	1108381	59	60	60	52	40	0.58	13.3	19.6	8.4	ABSENT	NOT DONE	NO ABNORMAL GENE FOUND	NOT DONE	-	-	-	-
57	851649	56	60	60	54	42	0.44	14.4	22	21	ABSENT	NOT DONE	MYH7	NOT DONE	-	-	-	-
58	382547	58	60	60	51	40	0.61	19.2	23.2	23.1	ABSENT	NOT DONE	NO ABNORMAL GENE FOUND	NOT DONE	-	-	-	-
59	1047867	62	60	60	53	58	0.59	16.2	23.4	24	ABSENT	NOT DONE	MYBPC3	NOT DONE	-	-	-	-
60	386825	54	60	60	58	54	0.46	14.5	23	21	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
61	1076244	58	60	60	56	58	0.44	13.4	19.4	20	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
62	1082791	52	60	60	51	48	0.49	16.4	22	22	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
63	221596	60	60	60	53	52	0.52	11	18.2	22	ABSENT	NOT DONE	MYH7	NOT DONE	-	-	-	-
64	1011493	58	60	60	52	56	0.55	18.4	20.4	24	ABSENT	NOT DONE	NO ABNORMAL GENE FOUND	NOT DONE	-	-	-	-
65	1083276	52	60	60	53	58	0.49	14.2	20.2	21	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
66	1108381	52	60	60	60	52	0.61	15.4	18.6	22	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
67	1087012	49	60	60	52	51	0.52	13.4	16.4	21	ABSENT	NOT DONE	NO ABNORMAL GENE FOUND	NOT DONE	-	-	-	-
68	1094704	53	48	45	42	49	0.44	10.9	13.1	24	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
69	1052079	56	60	60	55	54	0.49	15.5	16.2	22	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
70	1053705	58	60	60	53	54	0.56	13.2	14.9	19	YES	NOT DONE	MYBPC3	NOT DONE	-	-	-	-
71	591665	73	60	60	56	40	0.58	21.3	22.6	26	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
72	34475	42	60	60	38	35	0.65	9.9	15.3	25	ABSENT	NOT DONE	NO ABNORMAL GENE FOUND	NOT DONE	-	-	-	-
73	41406	62	60	60	53	42	0.5	11.4	13.4	21.1	ABSENT	NOT DONE	MYBPC3	NOT DONE	-	-	-	-
74	777590	56	60	60	51	55	0.58	12.5	14.3	22	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
75	875932	52	60	60	58	57	0.49	13.8	18.9	19	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
76	963403	51	60	60	55	42	0.52	13.8	16.4	20	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
77	41406	56	60	60	54	36	0.57	15.4	17.1	22	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
78	1114348	56	60	60	53	42	0.5	11.2	12.5	20	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
79	442020	51	60	60	50	58	0.43	13.2	16.5	22	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
80	986034	54	60	60	49	44	0.53	16.2	18.9	16	ABSENT	NOT DONE	NO ABNORMAL GENE FOUND	NOT DONE	-	-	-	-
81	991739	56	60	60	56	54	0.49	18	19.6	17	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-

Sr. No	MRD No.	Age	Sex	Chief Complaints					Diagnosis	Type	Past History							Personal History				Family History	Examination				Systemic Examination		
				Chest pain	SOB	Palpitations	Syncope	Presyncope			HTN	Bronchial Asthma	Arrythmia	CVA	DM	Heart failure	AICD implantation	Smoker/ Non Smoker	Alcoholic/ Non-Alcoholic	OSA	Marathon runner	SCD	Oedema	Pulse (/min)	SBP (mm Hg)	DBP (mm Hg)	Auscultation ESM – Rest/ Dynamic	Axis (degree)	Axis group
82	4698	17	M	NO	YES	NO	NO	NO	HCMP	APICAL	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	YES	NO	84	130	70	NO	30	Normal
83	484383	60	M	YES	NO	NO	NO	NO	HCMP	MID AND APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	72	130	80	NO	10	Normal
84	983863	58	M	NO	YES	NO	NO	NO	HCMP	APICAL	YES	NO	NO	NO	YES	NO	NO	NON	NON	NO	NO	NO	NO	84	140	80	NO	-30	Normal
85	979882	55	M	NO	YES	NO	NO	NO	HCMP	APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	80	130	80	NO	-45	Left
86	267515	28	F	NO	YES	NO	NO	NO	HCMP	REVERSE CURVATURE	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	YES	NO	84	130	80	YES	-30	Normal
87	1011304	64	M	NO	YES	NO	NO	NO	HCMP	APICAL	YES	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	NO	NO	72	140	90	NO	45	Normal
88	986006	67	M	NO	YES	NO	NO	NO	HCMP	MID AND APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	72	140	80	NO	-30	Normal
89	994102	52	M	NO	YES	NO	NO	NO	HCMP	APICAL	YES	NO	NO	NO	NO	NO	NO	NON	YES	NO	NO	YES	NO	81	130	80	NO	30	Normal
90	829204	54	F	NO	YES	NO	NO	NO	HCMP	MID AND APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	YES	NO	NO	NO	81	140	90	NO	-10	Normal
91	1149359	79	F	NO	YES	NO	NO	NO	HCMP	APICAL	YES	NO	NO	NO	YES	NO	NO	NON	NON	NO	NO	NO	NO	76	150	90	NO	-30	Normal
92	183340	54	F	NO	YES	NO	NO	NO	HCMP	APICAL	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	YES	NO	68	120	70	NO	10	Normal
93	1156535	77	M	NO	YES	NO	NO	NO	HCMP	APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	85	130	70	NO	0	Normal
94	1176731	42	M	NO	YES	NO	NO	NO	HCMP	MID AND APICAL	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	75	120	70	NO	30	Normal
95	294053	59	M	NO	YES	NO	NO	NO	HCMP	APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	80	140	90	NO	10	Normal
96	1143196	73	F	NO	YES	NO	NO	NO	HCMP	SIGMOID	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	YES	YES	102	110	70	YES	-45	Left
97	324139	69	M	NO	YES	NO	NO	NO	HCMP	MID AND APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	85	140	90	NO	10	Normal
98	1133075	58	M	NO	YES	NO	NO	NO	HCMP	NEUTRAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	73	140	80	NO	0	Normal
99	1139781	80	M	NO	YES	NO	NO	NO	HCMP	APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	75	150	90	NO	30	Normal
100	1156535	77	M	NO	YES	NO	NO	NO	HCMP	MID AND APICAL	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	78	140	80	NO	10	Normal
101	1198562	38	M	NO	YES	NO	NO	NO	HCMP	REVERSE CURVATURE	NO	NO	NO	NO	NO	NO	NO	NON	YES	NO	NO	YES	NO	79	118	76	NO	-20	Normal
102	968526	65	F	YES	YES	NO	NO	NO	HCMP	REVERSE CURVATURE	YES	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	YES	89	150	90	YES	-10	Normal
103	182437	42	M	NO	YES	NO	NO	NO	HCMP	APICAL	NO	NO	NO	NO	NO	NO	NO	NON	NON	NO	NO	NO	NO	73	120	80	NO	-60	Left

Sr. No	MRD No.	12 lead ECG						Echocardiographic Parameters																	
		Rhythm	PR interval (ms)	QRS duration (ms)	LVH	QT_c (Bazett's) (ms)	Giant T wave inversion	Maximum wall thickness (mm)	Opposing wall ratio	LVID-S (mm)	LVID-D (mm)	LVPW-S (mm)	LVPW-D (mm)	IVS-S (mm)	IVS-D (mm)	LVOT gradient: resting (mm of Hg)	LVOT gradient: valsalva (mm of Hg)	MR	SAM	Mitral valve-E/A	E/e'	IVRT (ms)	DT (ms)	PASP (mm of Hg)	LAVI (ml/m2)
82	4698	NSR	160	120	-	472	NO	21	-	34	52	16	14	20	18	-	-	NO	-	2.2	14	77	167	28	35
83	484383	NSR	160	120	-	443	NO	23	1.90	25	37	13	12	24	23	-	-	NO	-	1.6	18	84	172	25	36
84	983863	NSR	160	120	YES	468	YES	20	-	34	52	16	14	14	13	-	-	NO	-	1.3	13	67	153	22	33
85	979882	NSR	160	120	YES	442	YES	26	-	28	44	13	10	12	11	-	-	NO	-	1.5	16	79	165	30	32
86	267515	NSR	180	120	YES	462	NO	30	3.75	25	39	12	8	30	28	60	-	YES, MODERATE	YES	0.9	16	84	142	35	38
87	1011304	NSR	160	110	-	463	NO	27	-	28	48	13	12	27	25	-	-	NO	-	2.5	24	82	192	55	33
88	986006	NSR	160	120	YES	483	YES	30	2.00	29	50	16	15	30	28	-	-	NO	-	0.7	32	78	162	32	35
89	994102	NSR	160	120	YES	462	YES	21	-	29	45	14	10	12	11	-	-	YES	-	0.5	12	63	160	35	34
90	829204	NSR	160	120	-	456	NO	21	3.00	21	40	10	7	25	21	-	-	NO	-	1.5	11	98	165	20	32
91	1149359	NSR	160	120	YES	442	YES	18	-	30	44	10	7	13	10	-	-	NO	-	1.6	14.2	98	156	32	30
92	183340	NSR	180	120	-	460	YES	21	-	29	40	11	8	14	9	-	-	YES	-	2.3	20	42	230	20	44
93	1156535	NSR	160	120	-	452	YES	26	-	26	40	15	14	27	26	-	-	NO	-	1.2	12	63	198	40	42
94	1176731	NSR	160	110	-	463	NO	24	2.18	34	49	13	11	13	12	-	-	NO	-	1.2	16	89	156	20	28
95	294053	NSR	160	110	-	453	NO	25	-	35	50	16	12	16	13	-	-	YES	-	1	24	63	157	20	25
96	1143196	NSR	180	120	-	478	NO	22	1.29	20	31	20	17	22	20	45	-	YES, MODERATE	YES	2.5	26	98	146	30	35
97	324139	NSR	160	120	-	460	YES	28	3.50	34	51	11	8	12	9	-	-	YES	-	1.8	18	96	184	48	47
98	1133075	NSR	160	120	-	458	NO	20	2.50	30	43	10	8	20	16	-	-	YES	-	0.7	17	78	196	36	42
99	1139781	NSR	180	110	-	460	YES	20	-	34	56	13	8	13	12	-	-	NO	-	0.9	15	79	158	20	37
100	1156535	NSR	160	120	YES	462	YES	26	1.85	26	40	15	14	27	26	-	-	NO	-	1	12	80	163	40	42
101	1198562	NSR	160	120	-	452	NO	28	2.00	31	52	14	12	28	20	-	-	NO	-	1.4	10	98	183	30	34
102	968526	NSR	180	110	-	463	NO	32	3.00	34	59	11	9	32	24	50	-	YES, MODERATE	YES	1.9	22	88	193	50	36
103	182437	NSR	160	110	YES	438	YES	20	-	30	45	13	10	13	11	-	-	NO	-	0.8	12	68	148	40	32

Sr. No	MRD No.											24 hours Holter study (if required)	Genetic analysis (if required)	Radiological investigations	Cardiac MRI			
		LAEF= (MaxV-MinV) /MaxV%	EF by 2D echo (%)	EF by 3D echo (%)	Strain derived EF (%)	Stroke volume (ml)	LV MPI	GLS (-%)	GCS (-%)	LA strain (peak) %	Mid-cavity gradient				Ventricular thickness	SAM	LVOT obstruction	Variants of HCM
82	4698	51	60	60	51	60	0.48	18	19.6	19	ABSENT	NOT DONE	MYBPC3	NOT DONE	-	-	-	-
83	484383	65	60	60	52	42	0.46	16.5	18.3	18	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
84	983863	49	60	60	55	57	0.57	18	17.6	21	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
85	979882	53	60	60	50	46	0.59	10	11	21	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
86	267515	52	60	60	56	40	0.61	15	16.7	20	ABSENT	NOT DONE	MYH7	NOT DONE	-	-	-	-
87	1011304	51	60	60	52	52	0.58	17	16.2	19	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
88	986006	51	60	60	53	48	0.52	17	21.1	23	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
89	994102	51	60	60	52	52	0.49	18	16.5	21	ABSENT	NOT DONE	NO ABNORMAL GENE FOUND	NOT DONE	-	-	-	-
90	829204	58	60	60	51	55	0.36	22.1	20.6	22	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
91	1149359	52	60	60	53	53	0.44	22.2	19.9	19	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
92	183340	53	60	60	52	59	0.42	20.4	19.7	21	ABSENT	NOT DONE	NO ABNORMAL GENE FOUND	NOT DONE	-	-	-	-
93	1156535	48	60	60	52	55	0.44	22.1	20.8	22	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
94	1176731	58	60	60	59	59	0.39	10.4	9.4	20.5	YES	NOT DONE	MYBPC3	NOT DONE	-	-	-	-
95	294053	62	60	60	55	58	0.4	16.2	19.2	21.5	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
96	1143196	60	60	60	59	56	0.52	14.2	13.5	22	ABSENT	NOT DONE	MYH7	NOT DONE	-	-	-	-
97	324139	58	60	60	58	51	0.49	15.6	18.4	19	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
98	1133075	50	60	60	52	55	0.42	14.6	13.4	22	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
99	1139781	60	60	60	53	58	0.45	15.6	13.5	21	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
100	1156535	58	60	60	56	52	0.42	16.2	14.3	20.4	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
101	1198562	55	60	60	54	58	0.49	18.9	16.4	23	ABSENT	NOT DONE	MYH7	NOT DONE	-	-	-	-
102	968526	59	60	60	52	45	0.51	18.4	16.7	21	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-
103	182437	61	60	60	58	58	0.4	19.4	18.4	22	ABSENT	NOT DONE	NOT DONE	NOT DONE	-	-	-	-

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Date 11/5/2021

The Principal,
DMC, Ludhiana

Subject: Approval of Plan of Thesis.

This is to inform you that the Faculty of Medical Sciences in its meeting held on 22.01.2021 has approved the plan of thesis of Dr Anshuman Gupta, a student of your college for DM Cardiology along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis
Clinical and echocardiographic profile of hypertrophic cardiomyopathy

Supervisor
Dr. Shibba Takkar Chhabra
Prof.,
Dept. of Cardiology,
DMCH, Ludhiana.

Co-Supervisor
Dr. Abhishek Goyal
Assoc. Prof.,
Dept. of Cardiology,
DMCH, Ludhiana.

The student has to submit the three copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

2. *If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.*

CLINICAL AND ECHOCARDIOGRAPHIC PROFILE OF HYPERTROPHIC CARDIOMYOPATHY

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

DOCTORATE OF MEDICINE

IN

CARDIOLOGY

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES

FARIDKOT

MARCH, 2020

DR. ANSHUMAN GUPTA

DEPARTMENT OF CARDIOLOGY

DAYANAND MEDICAL COLLEGE & HOSPITAL

LUDHIANA

ABSTRACT OF PLAN OF THESIS

Title	CLINICAL AND ECHOCARDIOGRAPHIC PROFILE OF HYPERTROPHIC CARDIOMYOPATHY
For the degree of	D.M Cardiology
Name of the candidate	Dr. Anshuman Gupta
Supervisor	Dr. Shibba Takkar Chhabra, Professor, Department of Cardiology, DMCH, Ludhiana
Co-supervisor	Dr. Abhishek Goyal, Associate Professor, Department of Cardiology, DMCH, Ludhiana
Institution	Dayanand Medical College & Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: Hypertrophic cardiomyopathy (HCM) is an autosomal dominant disease characterised by the thickening of the left/right ventricle along with the interventricular septum. The Indian population exhibits a great range of geographic and genetic diversity in comparison to other populations; therefore there is need to examine the epidemiological and clinical presentation based on the gender specific differences, presenting complaints, age at presentation, family history.

Aim of Study: 1. To study the clinical and demographical profile of Indian Hypertrophic cardiomyopathy patients.

2. To compare echocardiographic characteristics with respect to subtypes of Hypertrophic cardiomyopathy in Indian patients.

Materials & Methods: This prospective and retrospective, observational and non-blinded study will be conducted to know the demographical and clinical profile of Indian Hypertrophic cardiomyopathy patients. All patients who already presented between 1st March 2019 to 28th Feb 2020 and all follow up and new patient's presenting to DMCH Ludhiana between 1st March 2020 to 31st May 2021 will be enrolled for study.

Clinical Significance: To see the clinical significance related to various subtypes of HCM with respect to age, sex, gender, NYHA grade, h/o diabetes, h/o smoking and alcohol and their correlation with them in Indian population.

Keywords: HCM, demography, echocardiography, clinical features.

(Signature)	(Signature)	(Signature)	(Signature)
Candidate	Co-supervisor	Supervisor	Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"CLINICAL AND ECHOCARDIOGRAPHIC PROFILE OF HYPERTROPHIC CARDIOMYOPATHY"** exist in the Department of Cardiology, DMC&H, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis is genuine and is collected by the candidate himself/herself under our supervision and guidance.

Supervisor

Dr. Shibba Takkar Chhabra
Professor
Department of Cardiology
DMCH, Ludhiana

Co-Supervisors

Dr. Abhishek Goyal
Associate Professor
Department of Cardiology
DMCH, Ludhiana

Head of the Department

Dr. Gurpreet Singh Wander
Professor & Head
Department of Cardiology
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Place : Ludhiana

Date : 1/5/20

APPROVAL PROFORMA
BY RESEARCH & ETHICAL COMMITTEE
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

Name of candidate	Dr. Anshuman Gupta
Department	Cardiology
Topic of Thesis	CLINICAL AND ECHOCARDIOGRAPHIC PROFILE OF HYPERTROPHIC CARDIOMYOPATHY
Likely date of appearing in PG examination	MAY, 2022
Date of enrollment	10th September 2019
Name of Head of Department	Dr. Gurpreet Singh Wander
1) Supervisor	Dr. Shibba Takkar Chhabra
2) Co-Supervisor(s)	Dr. Abhishek Goyal
Signature of Members of Research Committee with Stamp	**Signature of Members of Ethics Committee with Stamp**
Dr. Jaspal Singh (Professor & Head, Surgery)	Mr. A K Jindal (Member, Ethics Committee)
Dr. Dinesh Gupta (Professor & Head, Medicine)	Dr. Anu Sharma (Professor, Anatomy)
Dr. Sandeep Kaushal (Assistant Dean Academics)	Dr. Deepinder K Chhina (Professor & Head, Microbiology)
Dr. Rajoo Singh Chhina (Dean Academics)	Dr. Neena Sood (Professor & Head, Pathology)
Dr. Gurpreet S Wander (Vice Principal)	Dr. Gurpreet S Wander (Vice Principal)
	Dr. Gagandeep Singh (Secretary, Ethics Committee Professor & Head, Neurology)

Approved: Yes / ~~No~~

Dr. Sandeep Puri
Chairperson
Research Committee
Chairperson
Research Committee

Approved: Yes / ~~No~~

Prof. Arvind Malhotra
Chairperson
Ethics Committee
Chairperson
Ethics Committee

BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT

PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

1	NAME OF THE CANDIDATE AND ADDRESS	Dr. Anshuman Gupta H. No 1074, Ward no 5, Garcha street Basti Gobindgarh, G.T. Road, Moga
2	NAME OF THE INSTITUTE	DAYANAND MEDICAL COLLEGE AND HOSPITAL LUDHIANA
3	COURSE OF STUDY AND SUBJECT	DM CARDIOLOGY
4	DATE OF ADMISSION OF COURSE	10th September 2019
5	TITLE OF THE TOPIC	CLINICAL AND ECHOCARDIOGRAPHIC PROFILE OF HYPERTROPHIC CARDIOMYOPATHY

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

HCM is characterized by a thickened but non-dilated left ventricle in the absence of another cardiac or systemic condition (e.g., aortic valve stenosis, systemic hypertension, and some expressions of physiologic athlete's heart) capable of producing the magnitude of left ventricular (LV) hypertrophy evident.[1-4]

Hypertrophic cardiomyopathy (HCM) is a complex muscular disorder of the heart characterized by left and/or right ventricular asymmetric hypertrophy involving the interventricular septum. It is inherited as an autosomal dominant disease in at least 50% of the cases, though sporadic cases due to de novo mutations are also observed.

The condition is characterized by a heterogeneous disease expression with the prevalence reported to be 1 in 500 of the population.[5-6] The first clinical description of HCM was given in 1958 by Teare, who reported the sudden death in young patients.[7]

HCM Variants:

1. Midcavity obstructive HCM [8]
2. Midcavity obstruction with LV apical aneurysm [9]
3. Apical HCM [10]
4. Right ventricular obstruction
5. Obstructive HCM in elderly adults: Sigmoid septal morphology [11]
6. Transition to end-stage HCM [12]

Pathological features of HCM are: cardiac myocyte hypertrophy and disarray, interstitial fibrosis and thickening of intra-mural coronary arteries. Myocyte disarray is considered to be the pathological hallmark of HCM.

HCM exhibits great clinical variability from being asymptomatic to severe heart failure with the manifestation of the disease in the 4th/ 5th decade of life. The phenotypic variability can be attributed to compound heterozygosity, modifier genes, epigenetic factors (DNA methylation and imprinting), epistasis (interaction between genes), post-transcriptional and post-translational modifications of gene products, presence of coexisting diseases, and environmental influences.[13]

HCM has been widely studied, and a range of population based approaches have assessed the prevalence and incidence of HCM. The estimates varied almost by ten-fold which could be attributed to ethnicity, differences in populations, study methods and settings.

The studies showed a consistent male preponderance compared to females at all ages below 70 years, by a factor of two to three.[14] The epidemiology and natural history of HCM is incompletely understood, particularly with respect to the risk of sudden death. Epidemiological studies based on tertiary referral centers report higher deaths than community based studies.

Apart from the mutations in sarcomeric and cytoskeletal genes implicated in the manifestation of the disease, gender specific differences also contribute to the variable expressivity of the disease.

The disease exhibits extreme variability, in terms of age of onset, disease progression, occurrence of sudden cardiac death (SCD), spectrum and extent of symptoms, and most noticeably, the degree, and location of hypertrophy.[15-18]

6.2 REVIEW OF LITERATURE

A study was conducted by Advithi Rangaraju et al in 2014 which revealed 37% of sporadic cases and 63% of familial HCM cases, with a sex ratio of 3.1:1, indicating a male preponderance. The mean age at onset of 38 years was observed irrespective of the gender. Interestingly, males were predominant in the age group of 21-40 years exhibiting non-obstructive type of HCM while female preponderance was observed in the age group of 0-20 years and 40-60 years with high incidence of obstructive HCM cases associated with severe clinical manifestations and poor prognosis, based on the significant echocardiographic parameters. This was further substantiated with an early age at onset in females with asymmetric septal type of hypertrophy and in males with mid-cavity concentric hypertrophy enlightening on a strong genetic predisposition. Apical and mid-cavity concentric HCM were reported to be rare in this study, hence it is observed that gender, age at onset and type of HCM could contribute as epidemiological variables in the disease onset and could account for the heterogeneity with emphasis being laid on the genetic basis and established mode of inheritance in Indian population.[19]

A study was conducted by Amitabh Biswas et al in 2015, there were 59 patients with a clinical diagnosis of HCM with a preponderance of disease in males with a ratio (men, women) of 5.5:1. Average age of onset of the disease was late 30 s (39.2 ± 14.5) with familial HCM accounting for 18% (n = 9) for total HCM families (n = 50). Nonobstructive kind of HCM was more prevalent as compared to obstructive HCM (66.1% vs. 33.9%). Average posterior wall LV thickness of the HCM patients was 16 ± 4.8 mm and IVS thickness was 21 ± 8.3 mm with familial patients having greater wall thickness as compared to sporadic patients. Sequencing of hotspot region of MYH7 identified three mutations in three different patients in Indian population.[20]

A study was conducted by Ali J. Marian et al in 2017 where mutations in over a dozen genes encoding sarcomere-associated proteins cause HCM. MYH7 and MYBPC3, encoding β-myosin heavy chain and

myosin-binding protein C, respectively, are the 2 most common genes involved, together accounting for ≈50% of the HCM families. In ≈40% of HCM patients, the causal genes remain to be identified.[21]

A study was conducted by Qin Luo et al in 2020 where a total of 90 HCM patients with HTN and 172 patients without HTN were divided into a hypertensive group and non-hypertensive group. The clinical characteristics, cardiac structure and function, and prognosis of the two groups were compared and found that HCM patients with HTN had fewer syncope events in their medical histories (8% vs. 22%, $P < 0.01$) and sudden deaths in the family (3% vs. 10%, $P < 0.05$). The prevalence of apical hypertrophy (18% vs. 7%, $P < 0.01$) and midventricular obstruction (26% vs. 15%, $P < 0.05$) was higher in the HTN group.[22]

6.3 AIMS AND OBJECTIVES OF THE STUDY

- To study the clinical and demographical profile of Hypertrophic cardiomyopathy in our patients.
- To compare echocardiographic characteristics with respect to subtypes of Hypertrophic cardiomyopathy in our patients.

7.0 MATERIALS AND METHODS

This prospective and retrospective, observational and non-blinded study will be conducted to know the demographical and clinical profile of Indian Hypertrophic cardiomyopathy patients. All patients who already presented between 1st March 2019 to 28th Feb 2020 and all follow up and new patient's presenting to DMCH Ludhiana between 1st March 2020 to 31st May 2021 will be enrolled for study.

7.1 SOURCE OF DATA:

Data collection will be done by analyzing the patients with clinical features of HCM and confirmation on echocardiography.

7.2 METHOD OF COLLECTION OF DATA

All enrolled patients will undergo:

1. Ecg
2. Echo
3. BNP levels (if available)
4. Holter (if indicated)
5. Routine haemogram

Family members will be screened for HCM by ECG and Echocardiography. The American College of Cardiology (ACC)/American Heart Association (AHA) consensus guidelines recommend 12 years as the starting age for family screening of first-degree relatives of affected probands with hypertrophic cardiomyopathy (HCM).

Inclusion Criteria

Any patient who present to DMCH/HDHI in patient/ out patient clinic who have clinical and echocardiographic diagnosis of HCM

Exclusion criteria

1. Aortic valve stenosis
2. Physiologic athlete's heart

	HCM	ATHLETE'S HEART
Focal pattern of Lv hypertrohphy	Present	Absent
Lv cavity <45mm	Present	Absent

Lv cavity >55mm	Absent	Present
Left atrium enlargement	Present	Absent
Bizzare ecg patterns	Present	Present
Abnormal Lv filling pattern	Present	Absent
Family history	Present	Absent
Late gadolinium enhancement	Present	Absent
Pathogenic sarcomere mutation	Present	Absent
VO_2 Increase >110%	Absent	Present

Proposed Clinical Family Screening Strategies

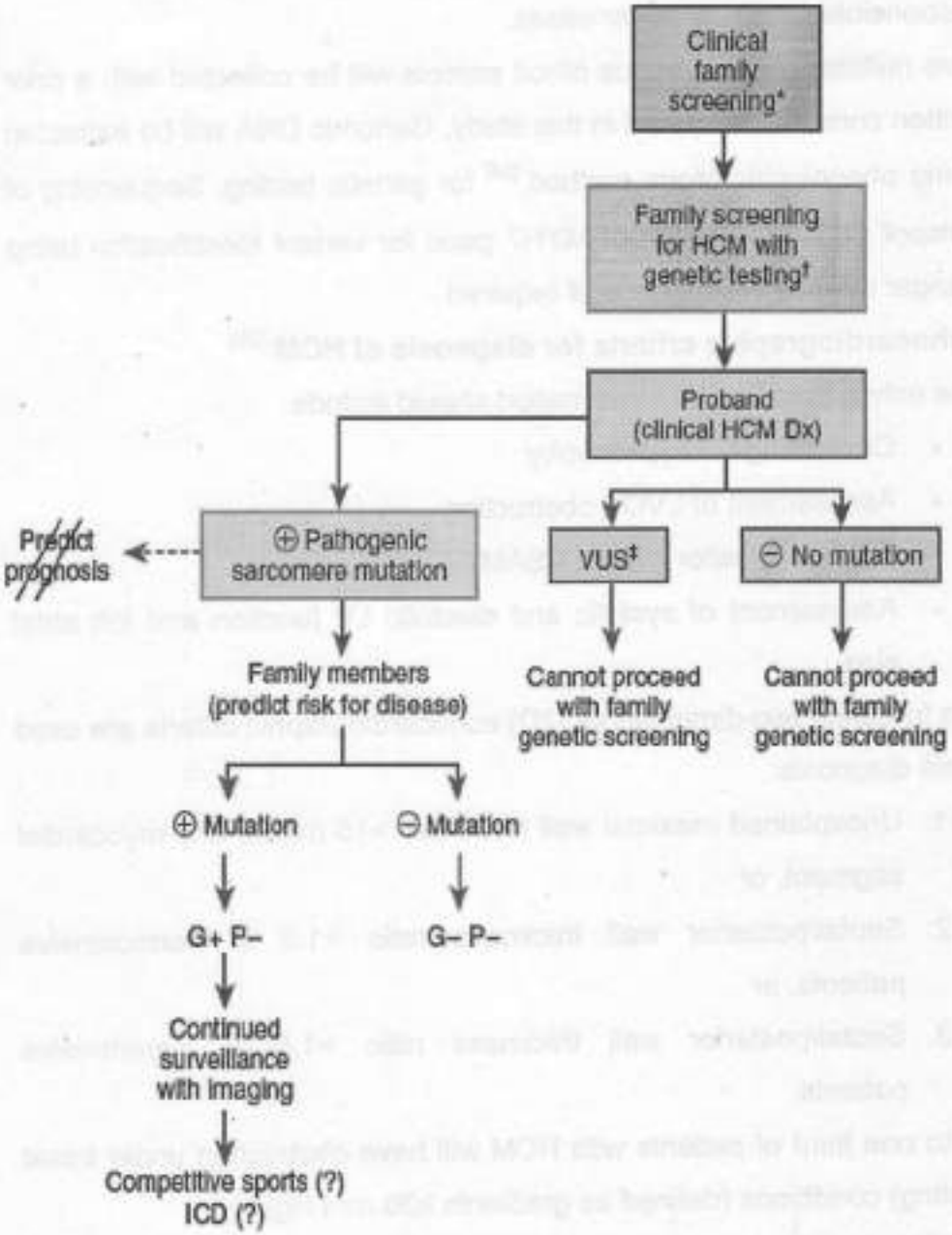
With Echocardiography or Cardiovascular Magnetic Resonance (and 12-Lead Electrocardiography) for Detection of HCM with Left Ventricular Hypertrophy [23]

Age <12 years : Optional unless:

1. Malignant family history of premature death from HCM, or other adverse complications
2. Competitive athlete in an intense training program
3. Onset of symptoms
4. Other clinical suspicion of early left ventricular hypertrophy

Age 12 to 21 years: Every 12 to 18 months

Age > 21 years: Imaging at onset of symptoms, or possibly at 5-year intervals at least through midlife; more frequent intervals for imaging are appropriate in families with malignant clinical course or history of late-onset HCM



Genetic Basis and Testing

HCM is transmitted as a mendelian trait with an autosomal dominant pattern of inheritance; every offspring of an affected relative has a 50% chance of inheriting the disease[1,2]

HCM is known to be caused by mutations in 11 or more genes encoding proteins of the thick and thin contractile myofilament components of the cardiac sarcomere or the adjacent Z-disc. Two sarcomere genes, β-myosin heavy chain (MYH7) and myosin-binding protein C (MYBPC3) are by far the most common, accounting for 70% of patients for whom genotyping has been successful. Troponin T

(TNNT2), troponin I (TNNI3), and several other genes are each responsible for 5% or fewer cases.

Five milliliter of intravenous blood sample will be collected with a prior written consent if required in this study. Genomic DNA will be extracted using phenol-chloroform method,[24] for genetic testing. Sequencing of hotspot region (exon 23) of MYH7 gene for variant identification using Sanger method will be done, if required.

Echocardiographic criteria for diagnosis of HCM [25]

The echocardiographic examination should include.

- Confirming LV hypertrophy
- Assessment of LVOT obstruction
- Systolic anterior motion - SAM,
- Assessment of systolic and diastolic LV function and left atrial size

The following two-dimensional (2D) echocardiographic criteria are used to aid diagnosis:

1. Unexplained maximal wall thickness >15 mm in any myocardial segment, or
2. Septal/posterior wall thickness ratio >1.3 in normotensive patients, or
3. Septal/posterior wall thickness ratio >1.5 in hypertensive patients.

Up to one third of patients with HCM will have obstruction under basal (resting) conditions (defined as gradients ≥30 mm Hg).

Another one third will have labile, physiologically provoked gradients (<30 mm Hg at rest and ≥30 mm Hg with physiologic provocation)[26]

The final one third will have the non-obstructive form of HCM (gradients <30 mm Hg at rest and with provocation). Marked gradients ≥50 mm Hg, either at rest or with provocation, represent the conventional threshold for surgical or percutaneous intervention if symptoms cannot be controlled with medications.

Echocardiographic predictors of unfavourable outcome

- Sudden death: Maximal wall thickness >30mm

- HCM related death: LVOT gradient at rest >30mmHg, EF <50%
- All-cause mortality: Left atrial diameter >48mm
- Heart failure development: LA volume >27ml/m2 at baseline of during follow up
- Intraventricular dyssynchrony: >45

Electrocardiographic features for diagnosis of HCM [27]

- Left atrial enlargement
- Left ventricular hypertrophy with associated ST segment / T-wave abnormalities
- Deep, narrow ("dagger-like") Q waves in the lateral > inferior leads
- Giant precordial T-wave inversions in apical HCM
- Signs of WPW (short PR, delta wave).

Dysrhythmias: atrial fibrillation, supraventricular tachycardias, PACs, PVCs, VT

OUTCOME: The prevalence of HCM, age and gender specific differences, various subtypes of HCM and there clinical impact would be known by this study.

STATISTICAL ANALYSIS: Data will be described in terms of range; mean ±standard deviation (± SD, frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations will be done using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

NO

7.4 HAS ETHICAL CLEARENCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

ATTACHED HEREWITH

8.0 LIST OF REFERENCES

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From M-Mode Echocardiography to CMR. JACC Cardiovasc Imaging. 2016;9:858.

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hypertrophic cardiomyopathy: The EPOCH-H study. J Pract Cardiovasc Sci. 2015;1:143-9.

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PROFORMA

Name: Sr. No

Age:

Sex:

MRD. No.

Address:

Chief complaints:

Chest pain (Canadian cardiovascular grading of angina)

Shortness of breath

Palpitations

Syncope

Presyncope

NYHA (I, II, III, IV)

Brief History of Present illness

Diagnosis-

Past History

- Hypertension
- Bronchial Asthma
- Ventricular tachycardia / Atrial fibrillation
- CVA
- Diabetes mellitus
- Heart failure

Personal History

- Smoker/Non Smoker:
- Alcoholic/Non-Alcoholic:

Signature of Candidate

Signature of Supervisor

Family History

Sudden cardiac death

Similar cardiac ailment (genetic basis)

Examination

GPE

Oedema :

Pulse :

Blood Pressure :

System Examination

-CVS: Inspection

Palpation

Percussion

Auscultation

12-lead ECG

1. Rhythm
2. PR interval
3. QRS duration
4. LvH : yes

   No
5. LBBB/RBBB
6. IVCD
7. QT (Bazett's formula)
8. Q wave
9. Giant T wave

Echocardiographic assessment

1. Maximum wall thickness (mm):
2. LVID- systole and diastole

Signature of Candidate

Signature of Supervisor

3. LV Posterior wall thickness- systole and diastole
4. IVS thickness- systole and diastole
5. LVOT gradient: resting and valsalva
6. MR : yes- 1+,2+,3+,4+

   No
7. SAM: yes

   No
8. Mitral valve- E/A, E/e', IVRT, DT
9. Tricuspid valve- PASP
10. Aortic valve
11. Opposing wall ratio
12. Left atrial enlargement:

    Left atrial volume index

Left atrial emptying fraction=(MaxV-MinV)/MaxV *100

13. Assessment of systolic function:

    EF (By 2D and 3D echo)

    Stroke volume

    Lv MPI(Myocardial performance index) by tissue Doppler

14. Strain –GLS and GCS
15. Intraventricular dyssynchrony

Holter study (if required)

Signature of Candidate

Signature of Supervisor

Genetic analysis(if required)

Radiological investigations

Cardiac MRI (if required):

a. Ventricular thickness
b. Mitral valve apparatus
c. SAM
d. LVOT obstruction
e. Variants of HCM
f. Aorta
g. Aortic valve

Others

Signature of Candidate

Signature of Supervisor

INFORMED PATIENT CONSENT

Date: _____

I.....D/o, W/o, S/o.....
am willing to participate in the study **"CLINICAL AND ECHOCARDIOGRAPHIC PROFILE OF HYPERTROPHIC CARDIOMYOPATHY"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

.....
Signature of the Witness

Name:

.....
Signature of the patient

Signature of Candidate

Signature of Supervisor

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य "**CLINICAL AND ECHOCARDIOGRAPHIC PROFILE OF HYPERTROPHIC CARDIOMYOPATHY**" में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

"CLINICAL AND ECHOCARDIOGRAPHIC PROFILE OF HYPERTROPHIC CARDIOMYOPATHY" ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

_____ _____

ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____ ਮਿਤੀ_____

ਪਤਾ _____ ਪਤਾ _____

_____

ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

_____

ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ